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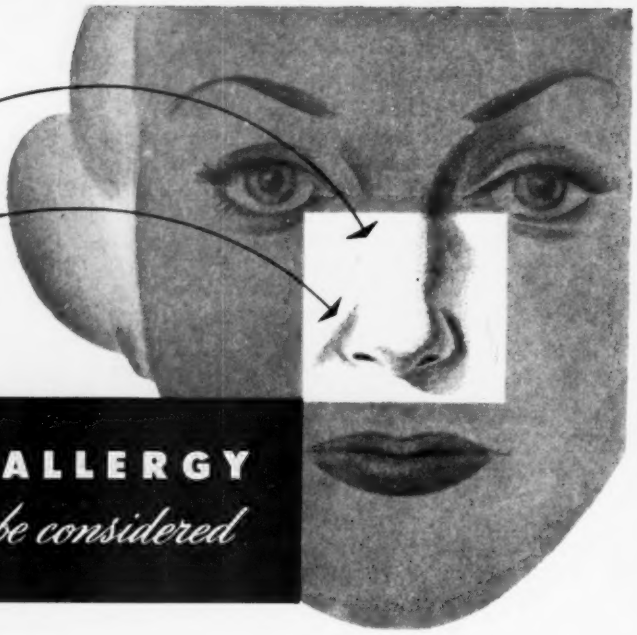

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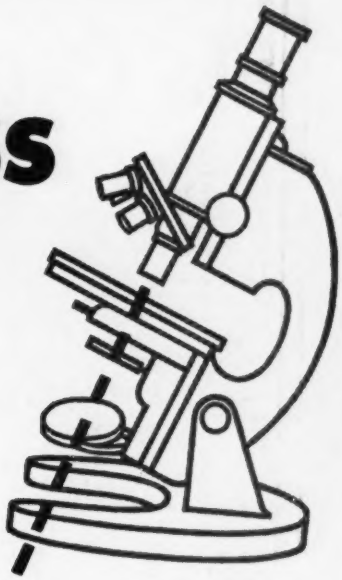

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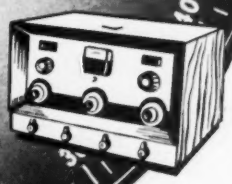

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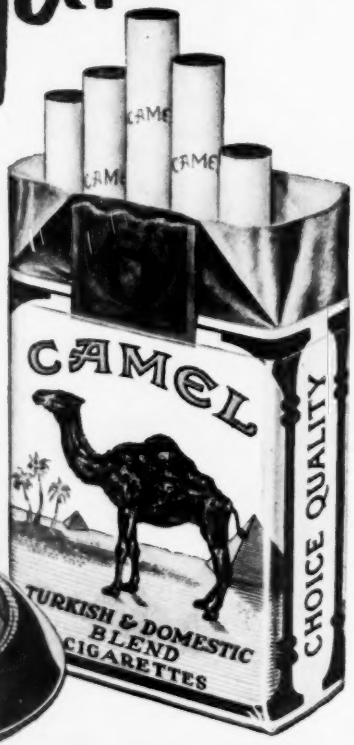

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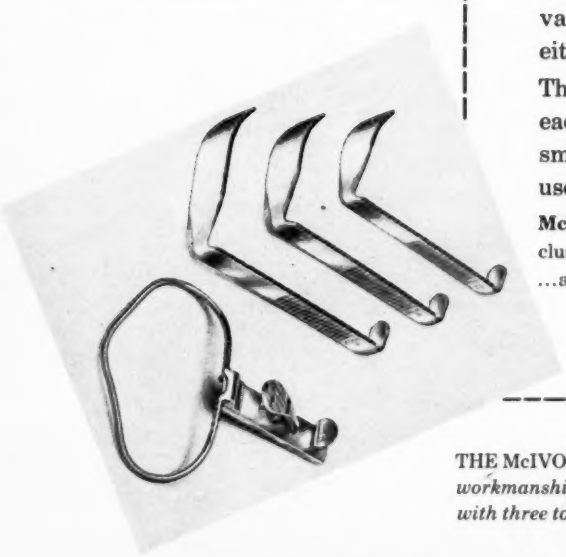

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VOLUME 55 MARCH 1952 NUMBER 3

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ALLERGY OF UPPER RESPIRATORY TRACT

Cure by Surgery Plus Antiallergic Methods

MARVIN FISHER JONES, M.D.
NEW YORK

PROBABLY the most important reason for presenting a paper on allergy of the upper respiratory tract is the need for continually alerting otolaryngologists to the frequency with which allergic symptoms occur in our specialty. Of 3,143 serial private case records reviewed for this paper, 522 gave positive evidence of allergy in some form. This incidence makes allergy an important item in my practice, as I believe it is in the practice of all otolaryngologists. All allergic symptoms should be scrupulously studied.

Repeated radical operations on the sinuses with less than satisfactory results were performed during a phase in the development of our specialty. This period of our development was sufficiently recent to remain fresh in our memory. Yet let us also remember that properly considered and efficiently performed surgery is still necessary to produce cures.

Allergic patients form a numerically increasing group for which nonsurgical or treatment methods were advised. Such treatment also has failed, in part, to produce an enjoyable symptom-free life.

In the past it was the custom for otolaryngologists to refer such patients to the allergists. Partly because of the allergist's lack of training in otolaryngological problems, the patients obtained less than satisfactory results. One exception to our rather disappointing experiences was the improvement obtained in cases of the seasonal allergies or hay fever. Even in this exception there was much to be desired. Skin tests are generally accepted as being reliable indicators of sensitivity for the seasonal varieties of allergy but perhaps as generally are considered poor indicators of food sensitivity. In our specialty the most confusing problems occur outside the seasonal allergy field.

How can we decide upon reasonable methods that give a promise of successful relief of symptoms for our allergic patients? An acceptable arrangement for the treatment of allergic patients is to combine the efforts of a competent allergist and an otolaryngologist on a parity basis. When a competent otolaryngologist will spend the necessary time to perfect himself in the technic of allergy treatment, his results will be most satisfactory.

Some otolaryngologists have preached the intelligent treatment of a patient as a whole. A specialist should be one with such ability plus additional and superior knowledge of a special part. With this approach, our object is to get the patient well in the shortest time by using all means at our disposal.

Read before the Section on Laryngology, Otology, and Rhinology at the One-Hundredth Annual Session of the American Medical Association, Atlantic City, June 13, 1951.

With this end in view the following tested program is proposed in which all procedures must be unstintingly followed: (*1*) daily physical outdoor-air exercise; (*2*) establishment of a controlled emotional balance in proportional living; (*3*) adjustment of damaging irregular living habits; (*4*) elimination of contacts with known allergic irritants; (*5*) desensitization to dust and molds and to histamine; (*6*) selection of a nutritionally complete, allergen-low diet; (*7*) application of skin tests with appropriate treatment for seasonal allergies; (*8*) administration of antihistamines for treatment and diagnosis when indicated; (*9*) correction of organic irregularities, and (*10*) doing the necessary surgery in carefully selected cases.

Quite by accident this is a 10-point program for rehabilitation of the ailing allergic patient. The time allotted will not permit a full consideration of all topics; therefore, I shall select the three methods that may be most controversial and less generally employed. Item 5 deals with desensitization by the minimum dose method. Item 6 concerns the selection of diets for food-sensitive patients, and the tenth subject is carefully considered surgical procedures.

Hansel [1] was probably the first to use the minimum dose method in the treatment of allergies. His excellent results were soon duplicated by Ashley,[2] who used similar methods. In rapid succession other otolaryngologists were able to use the minimum dose desensitization treatments with gratifying results. Many times the cure is spectacular. I have used Hansel's dust preparation combined with an extract of feed mill dust, similar to that reported on by Wittich, which consists almost entirely of molds. Histamine phosphate has proved to be satisfactory. My test dosage varies. The dilution is gauged by information gained in taking a most careful complete history. Patients who give evidence of having a marked sensitivity receive an intracutaneous test with 0.05 cc. of a 1 : 1,000,000,000 dilution. When there is an equal reaction to dust and to histamine, the diluent solutions are used as a control. The reaction does not determine whether or not the treatment shall be used; it merely guides in the selection of a desirable dilution with which to start treatment. A surprising fact is the frequency with which the test skin dose acts as an adequate therapeutic procedure. I have tried both intracutaneous and subcutaneous methods of administration. The intracutaneous initial dose followed by a subcutaneous injection has proved to be most satisfactory. The treatment starts with 0.05 cc. intracutaneously, and three days later 0.1 cc. is injected subcutaneously. The treatments are given twice a week, increasing 0.1 cc. with each dose until a dose of 0.5 cc. has been reached. If no improvement has occurred, I then repeat the process with the next stronger dilution. As an example, if I use 1 : 1,000,000,000 for my first series of five treatments, I use 1 : 100,000,000 for my next series. Further treatment is not administered when definite improvement is evident. Immediate improvement may occur after the first test dose. Should

1. Hansel, F. K.: Some Experience with Small Dosage Dust and Pollen Therapy, South. M. J. **38**:608-613, 1945.

2. Ashley, R. E.: Dust Therapy in Allergy of the Ear, Nose, and Throat, Ann. Otolaryng. **58**:417-428, 1949. I have used the methods advocated by Hansel and also used by Ashley with great success. The intradermal skin tests are done with a 1 : 1,000,000 dilution of Hansel's dust and a 1 : 1,000,000 dilution of histamine. Following their practice, I also use the skin tests to regulate dosage for hypersensitive patients rather than as a determination to use or not to use the treatments.

such an immediate improvement occur, further treatment is not considered. When improvement is unquestionably observed, it is human nature to want to give more of the same treatment in order to make sure of a lasting result. However, further therapy may injure the results gained. Again I wish to emphasize that it is a mistake to continue treatment after a satisfactory response has been obtained.

Diet is a most important part of an "all-out" treatment for the cure of allergy. Since skin testing for allergy to foods has proved to be not only useless but actually misleading, I prefer the clinical method of testing. Foods selected because of their low incidence as clinical disturbers are used by me for treatment and eventually for diagnosis. The selected foods seldom cause symptoms. Axiomatically, the foods which do commonly cause symptoms are therefore avoided. Unfortunately, the foods named in most lists as being allergic foods have been selected by skin testing. A list of foods is being compiled on the basis of clinical reaction. Time will be needed to prepare such a list. Most authorities agree on some foods. For example, the most frequent offenders are milk and milk products, wheat and wheat products, eggs, shellfish, chocolate, and nuts. Corn has been named by some as a common source of trouble, but, for some unexplained reason, corn has not been an important item in my experience. Unfortunately, the three foods whose desirable proteins are of greatest value are milk, eggs, and meat. Since milk and eggs come at the head of the list of allergic foods and since meat causes trouble less frequently, we choose meat with its natural fat as the basis of our diet. Pork and beef are the commonest symptom-producing foods, and if we suspect either one or both we may substitute fin fish or fowl for the test period. There is no actual nutritional deficiency in the diet recommended, but when children remain on the diet for a long time popular opinion makes calcium therapy almost mandatory. Vitamin B complex is low, as it is in most urban diets. Supplementary vitamins are sometimes ordered. Since the discussion thus far has been on the negative side, perhaps some positive suggestions are in order. Fat meat is consumed to capacity three times a day. The present vogue in breakfasts that calls for "a dash of orange juice, a weak piece of anemic toast and a shot of coffee" has obliterated the memory of ham and eggs with buckwheat cakes. This starvation breakfast fad must be physically and mentally degenerative. A substantial breakfast needs no apologies. Liver, kidneys, and sweetbreads are recommended for consumption more regularly than is the custom. Vegetables in this diet include sweet potatoes, raw carrots, beets, artichokes, brown rice, wild rice, and soy beans. The fruits include avocados, grapefruit, lemons, limes, dates, figs, prunes, apricots, pears, plums, and cherries. Fluids permitted are tea with lemon and sugar, lemonade, and water between meals. When the patient has been symptom-free for six weeks, a single food is added to the diet, and this added food is given to the point of toleration. Should the patient relapse into his previous ailing state, the added food is again removed. If there is a reaction following the second trial, the offending food is removed from the diet.

This method attempts to detect offending foods through clinical trial. The rice diet, milk diet, meat and potato diet, and other similarly severely restricted diets accomplish cures, since any such diet may eliminate personally offending foods. However, foods given in such concentrated excess without variety may sometimes sensitize patients to the ingested foods. A less Spartan diet can be prescribed, and this diet can be rapidly liberalized after the six-week trial period.

There is no denying that the answer to a dietary problem is obtained the hard way from the time standpoint. Occasionally, a year to a year and one-half elapses before a tolerated diet can be finally selected. However, the final results in restored health on a permanent basis are most gratifying.

Experience surely is a good teacher, but it is difficult to fit the lessons taught by experience into an I. B. M. machine. Questionnaires are becoming an increasing annoyance, and statistics derived therefrom can be misleading. To have integrity, a presentation should indicate the value of results obtained through years of experience. We have seen patients with diseases of the ear, nose, and throat cured by surgery; we have seen patients with symptoms of allergy cured by antiallergic treatment, but we have also seen patients who have received no benefits either from surgery or from allergy treatments when these methods were used alone. During the past years I have seen a significant number of patients who had not benefited through either method by itself become symptom-free after the intelligent concurrent use of surgery and antiallergic treatment. The types of surgery used include submucous resection, adenoidectomy, tonsillectomy, a variety of accessory sinus operations, surgery of the ear, and nasal polypectomy.

The five patients described in this paper complained of symptoms due to recurrent polypoid sinusitis. These patients include four adult males (one, a physician) and one maturing female. Those persons who are interested in the emotional theories regarding allergy will be furnished an opportunity to speculate. All these patients were legitimate candidates for radical sinus surgery. All of them would have needed bilateral pansinus operation. The results from such operations have been somewhat less than completely satisfactory as a permanent cure in my experience. My observations of the results of other surgeons have dampened my enthusiasm for extensive radical pansinus surgery. All patients had consulted other physicians who advised radical surgery, but the patients all refused to submit. The selection was made because of their present symptom-free status, a condition unknown to them previously. All had had both surgery and antiallergic treatment but had never received the two concurrently. They all had in common a few of the same complaints. Each patient had had "nasal trouble" all his remembered life. The main trouble was nasal obstruction. Other complaints were sneezing, watery or thick nasal and postnasal discharge, frequent colds at some time in their lives, a below-par lack of energy and tired feeling, poor tolerance for air conditioning and temperature changes. They all had allergic histories and repeated nasal operations. All patients had been subjected to surgical procedures alone without satisfactory results. Antiallergic treatments alone had also failed. The combination of essential surgery plus antiallergic treatments produced health satisfaction and symptomatic cure. The irreversible pathology of the upper respiratory tract did not, of course, return to normal.

REPORT OF CASES

CASE 1.—W. O'M., stated in a letter to his friend, "I have had a great deal of trouble with my sinuses for many years. I have taken about all the treatments, including 20 or more years of antiallergy injections, short-wave treatments, operations, penicillin vapor treatments, and a few I may have forgotten. None of them did me any permanent good." I did a polypectomy on this patient during May, 1948, plus desensitization to dust and histamine concurrently. He

had a second series of seven injections during the fall and early winter of 1948, and after that, he was pronounced cured. He reported back in January and in August, 1949, and then he was discharged.

CASE 2.—H. F. had a record of 10 polypectomies and 1 exenteration of the ethmoid and sphenoid areas. There were also other unrecorded removals of polyps in addition to those mentioned. In 1949 this patient had a polypectomy combined with nine dust and histamine injections. He reported back three times more, during November, 1949, and during March and May, 1950, and then he was discharged.

CASE 3.—A. B. is a physician. I have removed polyps from his nose every time he had a moment of spare time since 1929. He has had a long history of surgery. The last removal of polyps was combined with injections of dust and histamine. His upper respiratory tract is now in a condition that, for the first time, is satisfactory to us both.

CASE 4.—I. K. presented a similar history of lifelong polypectomies at frequent intervals. The last time his polyps were removed was in 1948. At that time he was also given the histamine and dust treatment. He is still under observation. One small polyp in his left side is being watched. This polyp fluctuates in size and at present has the size and contour of a very immature lima bean. We are both pleased with his symptomatic result.

CASE 5.—F. N., repeated removal of adenoid tissue and postnasal polyps had made this high school girl allergic to further surgery. She was given the selection diet plus desensitization with satisfactory results, but she was not cured. A fairly large postnasal polyp and hypertrophied adenoids were reduced in size during her treatment. She was able to breathe through her nose and ceased snoring for the first time in years. The former nasality in her voice improved. Because the result was less than we believed possible, the removal of the polyps and remaining adenoid tissue was recommended. A choanal-antral polyp of large size, together with small adenoid masses, was removed Jan. 3, 1951. The patient is, I believe, cured.

COMMENT

Five patients who had over many years received either surgical or antiallergic care alone without results have obtained symptomatic cure by combined polypectomy and antiallergic treatments.

ABSTRACT OF DISCUSSION

DR. IRVING W. VOORHEES, New York: There isn't much I can say because the subject has been so ably presented by my colleague and friend, Dr. Jones. I think, however, in the light of the increasing number of injections given by hypodermic needle for various diseases, we might well watch for an induced allergy through injections of different materials for various conditions. I have seen several such cases. One patient was a nurse in a hospital, and she wished to join the WAC's. She said that she had a very slight cold. She went to someone who injected vaccines into her skin subcutaneously, I found, instead of intradermally; the doses, she thought, were rather large, although she was a little uncertain about that. She thought about 3 cc. or perhaps 2.5 to 5 cc. was injected at each sitting. She had six such injections. After each one, her nose would swell almost entirely shut, a condition she had never had before. We desensitized her with the help of an allergist, and I, as the rhinologist, did whatever I could do for her. At the end of three weeks she telephoned to say that she had been accepted into the service. That may not be a scientific way to handle this matter. I simply want to call attention to the fact that it is possible.

DR. L. L. COLEMAN, New York: I should merely like to add to this discussion a very interesting case report. A patient was referred to me for his sixth polypectomy over a period of seven years. Prior to this, he had had a complete allergic study. After my polypectomy, I again suggested another allergic study, which was done by a very competent allergist. The patient was given a complete course of dust desensitization over a period of about two years. The results again were negative. Subsequent to that, he acquired a virus infection; for which his family physician gave him doses of aureomycin. Here we have a man who has had continuous nasal

allergy for a period of 10 years with no relief from any type of therapy. Aureomycin, however, gave him complete relief of all nasal symptoms. At the present time, on doses of 50 mg. of aureomycin four times a day, the patient is completely free of all symptoms of vasomotor rhinitis. He has maintained his symptom-free state for two years. When use of aureomycin is stopped, he again gets his nasal stuffiness and rhinorrhea and his polyps enlarge. When aureomycin is given again, his symptoms disappear.

I add this case to the previous discussion as evidence of the fact that in some cases nasal allergy unquestionably is infectious in origin.

DR. SACKS BRICKER, Philadelphia: My experience with these upper respiratory allergies leads me to conclude that the vast majority are of bacterial origin and that foods play a very small role in the upper respiratory forms. The polypoidal ethmoid diseases that we see are, in my opinion, allergic manifestations, and except for the pedunculated type of polyposis, I do a complete excision of the ethmoid capsule and very seldom get a recurrence of the polyps.

In many, many thousands of cases I have used the form of treatment outlined by the late Dr. George McKenzie, that of taking the nasal secretions, the oral secretions, and the secretions of the pharynx and making highly diluted filtrates and injecting them in weekly doses, sometimes at five-day intervals. Except for a few patients—I should say less than 1%—in which I should say the condition is so obstinate that they cannot get symptomatic cure, the results were good. This condition starts in children 1 or 2 years of age, some of whom have asthmatic symptoms, and it clears up almost uniformly after a few months of treatment. Naturally, when some surgery is indicated for obstructive lesions, it must be done. I don't trim the turbinates; I don't destroy any mucous membrane. If necessary, I do subcutaneous electrocoagulation.

To me the treatment of upper respiratory allergy is not a very difficult problem. I feel that if we would investigate Dr. McKenzie's philosophy a little more keenly, we would arrive at some very satisfactory results, which occasionally are dramatic after years and years of trouble.

DR. HENRY M. SCHEER, New York: The very fact that this subject has brought up so much discussion from the floor is an indication to us that Dr. Marvin Jones has brought before us a very timely and most interesting subject. There is no question that each and every one of us runs into rhinological problems, particularly rhinological problems that originate as a result of some allergic status. I am in agreement with Dr. Jones that these conditions form a good percentage of the cases encountered in our offices. He has outlined every factor from the standpoint of each of us becoming, because of our narrowed field in ear, nose, and throat research, somewhat of an allergist, and thus encroaching on someone else's territory. There is nothing that he hasn't covered. Dr. Jones has outlined the simple procedures that we, as rhinologists, should apply: that we should be physicians and study the dietetics of our patients; that we should be surgeons and know when and how much to operate when these indications are present. I think he has covered the subject admirably.

I rise to cite one particular instance which would probably fall in the category of one of the previous speakers and discussers who mentioned aureomycin. This patient was being treated for a chronic otitis media purulenta, a chronic discharging ear, and at the same time had a very profuse postnasal mucoid secretion. She would vomit incessantly in the morning, and because no specific pathology could be found she was treated most conservatively. At the time I saw her, certain home treatment had been advised. During the course of her treatments, she acquired a cystitis for which she went to a gynecologist. The gynecologist prescribed aureomycin. This aureomycin treatment lasted approximately five to six weeks, and during that time I saw her two or three times. The postnasal secretion—I observed the condition of the patient's ear periodically from the time she was 14 years old, and I saw no indication for surgery—during the six weeks spontaneously cleared up. When I say the secretion was profuse, I mean that one could use a suction bottle and take out a tablespoon or more of it from her pharynx each time.

So let's think about some of these incidental cures that occur, and maybe in certain instances, as Dr. Marvin Jones has mentioned, apply some of these after study, sometimes empirically.

DR. MARVIN F. JONES, New York: Of course, when anyone assigns himself such a topic, he knows that the subject is controversial, and he profits by the controversial arguments that are brought up.

My friend, Dr. Voorhees, mentioned injections. I am one of those persons who hates a needle, especially after I received injections every hour for about 10 weeks. The use of a needle, to me, is not a pleasant procedure.

I recall one of my patients, the physician I spoke of, who insisted he was so much better that he was going on a trip; in fact, he was going abroad and wanted, as a precautionary measure, to take some more of the same treatment that had cured him; so he took more medication, and he had a recurrence of his allergic symptoms. Even on the dosage that we give, you can lighten them up. There is every opportunity for a foreign protein to get in and cause the very thing that you are trying to cure. It is done every day, but it is usually caused by the larger dosage, and I think that is one of the failures, or one of the reasons for a failure.

Dr. Coleman brought up the subject of infection, and that would take a long time to answer; however I will say this, Dr. Coleman gave dust treatments to his patient over a period of two years, according to the statement. There is no patient in any of the series that I have had that has had any of the treatments over a period exceeding three weeks. I think long-term treatments defeat our purpose in this matter, and again I wish to remind you that we are dealing with a controversial subject. The infections that were mentioned were ones I deliberately avoided. If you have an infection, it is very normal to suppose that aureomycin will control that infection better than anything else. It is conceivable that you have a beginning allergy on which an infection is superimposed, and there is no reasonable reason why you shouldn't use antibiotics or sulfonamides to clear up that infection. That phase of it did not enter into the discussion of this paper.

Regarding Dr. Sacks Bricker's comments on the nasal secretion, I wish to remark that there is no doubt about the fact that there are certain nasal secretions that when the injections are done will promote cures. There is also the theory—and I believe a tenable one, although I don't believe it is the whole story—that the very bacterium itself acts as foreign protein and can cause a foreign protein reaction just as any other allergen can. I can't quite agree with Dr. Bricker that allergy is not a problem; certainly it is in my office, and it is one of the worst problems that I have. I wish that I were as fortunate as Dr. Bricker and could say that allergy presented no problem to me. It does, and it gives my patients trouble.

Dr. Scheer's use of aureomycin for treating profuse nasal discharge, I think, is the same type of therapy for treating an infection, but we must remember, as Dr. Robb very kindly advised me before I got to the platform, that the aureomycin occasionally causes a proctitis, a condition that is not always comfortable and can be permanent.

SYMPTOMS AND TREATMENT OF PITUITARY TUMORS

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HISTORY

PITUITARY derives its name from *pituita*—mucus—which, according to Galen [1] (end of second century, A.D.), was formed in the brain and discharged by this structure partly through the nose (catarrh). Thus the nasal cavity was called the cloaca of the brain (Zuckerkandl [2]). This theory was disproved only as late as 1655 by Schneider,[3] the same author who established the fact that the sinuses contain air and not mucus. Schneider explained that the cribriform bone (plate) has holes only as a dry bone, but that there is no free communication in the living organism between the brain and the nose. The other name, hypophysis (*hypo*—under, *physis*—growth), was given by von Soemmering [4] in 1798. Albrecht von Haller [5] (1708-1777) recognized two lobes in the pituitary body. A congenital anomaly was among the first disorders observed. Klinkosch,[6] in 1774, described an open canal in the sphenoidal bone and the pituitary body lying within this canal, visible in the oral cavity as a tumor the size of a hazelnut. One can assume that tumors of the pituitary body had been observed in autopsies even earlier. The first description of them are by Krimer [7] (1821) and by Rayer [8] (1823), who referred to similar observations as far back as 1734. Another case of pituitary tumor with amaurosis was mentioned by Ward [9] (1823). The embryology of the pituitary gland was described by Rathke [10] (1838).

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Amplification of a lecture given by invitation at the Annual Meeting of the Pacific Coast Oto-Ophthalmological Society, May 28, 1951, Victoria, B. C.

1. Galen, C.: De usu partium corporis humani, in Opera omnia, edited by C. G. Kuhn, Leipzig, 1882, Bk. 8, p. 650.

2. Zuckerkandl, E.: Normale und pathologische Anatomie der Nasenhöhle und ihrer pneumatischen Anhänge, Ed. 2, Vienna, W. Braumüller, 1893, Vol. 1, Chap. 1.

3. Schneider, C. V., cited by Zuckerkandl.[2]

4. von Soemmering, S. T.: De corporis humani fabrica, 1798, Vol. 4, No. 60, p. 69.

5. von Haller, A.: Elementa physiologiae corporis humani, Bern, 1757-1766; Prima linea physiologiae, 1747; First Line of Physiology, translated by W. Cullen, 1779, p. 168.

6. Klinkosch, J. T.: Dissertationes medicae selectiones Pragenses, 1775-1793, Vol. 1, No. 12; cited in Rathke, H.: Arch. Anat. Physiol. 1839, p. 232.

7. Krimer, W.: Seltene Veränderungen des Gehirnanhanges, J. prakt. Heilk. **52**:46, 1821.

8. Rayer, P.: Observations sur les maladies de l'appendice sus-sphenoidale (glande pituitaire) du cerveau, Arch. gén. méd. **3**:350, 1823.

9. Ward, J.: Case of Amaurosis Produced by Enlargement of the Pituitary Gland, London M. Reposit. **20**:217, 1823.

10. Rathke, H.: Über die Entstehung der glandula pituitaria, Arch. Anat. Physiol. **5**:482, 1838.

The diagnosis of a pituitary tumor or of a pituitary disorder during life was not made in this stage of medicine. The knowledge of characteristic bodily changes, of pathognomonic visual defects and of roentgen rays was not available. This diagnosis was anticipated by the publication of Pierre Marie [11] in 1886, describing acromegaly and recognizing it (1890) as due to a pituitary tumor; by Fröhlich [12] (1901), describing the adiposogenital type without acromegaly; by Oppenheim [13] (1899) calling attention to changes in roentgenography which have been studied in detail by Erdheim (1904) [14] in collaboration with Schüller and by Schüller (1905) [15]; and finally—not chronologically—by the discovery in 1874 of the semidecussation of the optic nerves (von Gudden).[16] Benda [17] (1900) and Erdheim [18] (1903) established the histological nature of the pituitary gland and classified tumors according to the three cell types. Erdheim [14] described in 1904 the hypophyseal-duct tumors, later called by Cushing [19] craniopharyngiomas. I [20] described the ophthalmic type (1912) produced by pituitary tumors, which was neither acromegalic nor adipose, with visual disturbances predominating. The discoveries of the physiological properties of the pituitary body, first of the posterior (Howell,[21] 1898), later of the anterior, lobe, aided the diagnosis of the disorders. Genital disturbances due to the pituitary tumors were observed by clinicians and surgeons in the beginning of this century. Zondek [22] clarified (1930) the role of the pituitary gland as the motor of

11. Marie, P.: Sur deux cas d'acromégalie; hypertrophie singulière non-congénitale des extrémités supérieures, inférieures et céphalique, Rev. méd. **6**:297, 1886; L'acromégalie, Nouv. icon. Salpétrière **1**:173, 1888; **2**:45, 96, 139, 188, 224, and 327, 1889; Brain **12**:59, 1889-1890; Sur deux types de déformation des mains dans l'acromégalie, Bull. mém. Soc. méd. hôp. Paris **13**:413, 1896. Marie, P., and Souza-Leite: L'acromégalie, Dissertation, 1890; cited by von Frankl-Hochwart, L.: Die Diagnostik der Hypophysis Tumoren ohne Akromegalie, Wien. med. Wchnschr. **59**:2127; 2257; 2326, 1909.

12. Fröhlich, A.: Ein Fall von Tumor der Hypophysis cerebri, Wien. klin. Rundschau **15**:883, 1901.

13. Oppenheim: Discussion, Berl. Gesellsch. Psychiat. u. Nervenkrankh. Nov. 13, 1899, Arch. Psychiat. **34**:303, 1901.

14. Erdheim, J.: Über Hypophysenganggeschwülste und Hirncholesteatome, Wien. Sitzungsb. math. u. naturwissensch., Classe 5, 113, 3, 1904.

15. Schüller, A.: Die Schädelbasis im Roentgenbilde, Fortschr. auf. Geb. Röntgenstrahlen, Ergänzungsband **2**:73, 1905.

16. von Gudden, B.: Über das Chiasma Nervorum opticorum, von Graefes Arch. Ophth. **20**:249, 1874; Über die Kreuzung der Nervenfasern im Chiasma Nervorum opticorum, ibid. **25**:1, 1879.

17. Benda, C.: Beiträge zur normalen und pathologischen Histologie der menschlichen Hypophysis cerebri Berl. klin. Wchnschr. **37**:1205, 1900; Über den normalen Bau und einige pathologischen Veränderungen der menschlichen Hypophysis cerebri, Arch. Anat. Physiol. **314**:373, 1900.

18. Erdheim, J.: Zur normalen und pathologischen Histologie der Glandula thyreoidea, parathyreoidea, und Hypophysis, Beitr. path. Anat. **33**:158, 1903.

19. Cushing, H.: The Chiasmal Syndrome of Primary Optic Atrophy and Bitemporal Field Defects in Adults with Normal Sella Turcica, Arch. Ophth. **3**:505 (May) 1930.

20. Hirsch, O.: Die operative Behandlung von Hypophysentumoren nach endonasalen Methoden, Arch. Laryng. u. Rhin. **26**:529, 1912.

21. Howell, W.: Physiological Effects of Extracts of Pituitary Body, J. Exper. Med. **3**:215, 245, 1898.

22. Zondek, B.: Die Hormone des Ovariums und des Hypophysenvorderlappens: Untersuchungen zur Biologie und Klinik der weiblichen Genitalfunktion, Berlin, Springer-Verlag, 1931.

the genital function; I [23] verified this in autopsied pituitary tumors (1931). The differentiation of pituitary tumors from other brain tumors, including hydrocephalus, was made possible by Dandy,[24] by contrast filling (Sgalitzer [25] and Schüller [26]) and by arteriography (Egas Moniz [27]). Parallel with the increase in diagnostic signs, there developed surgical and x-ray therapy.

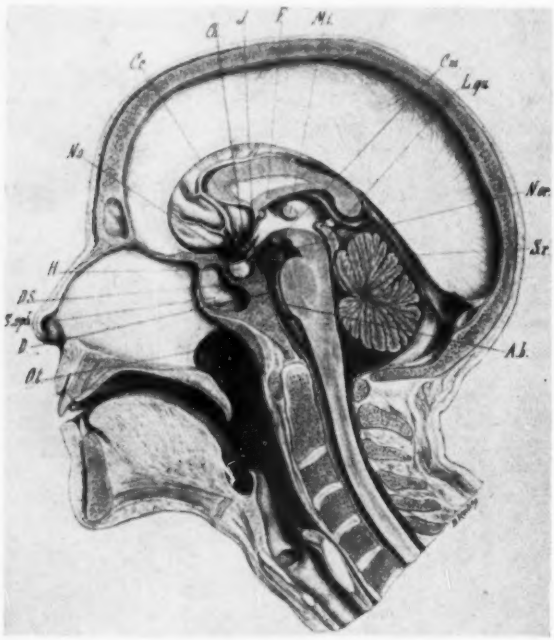

Fig. 1.—Sagittal section through the skull (F. Hochstetter, Institute of Anatomy, Vienna). In this drawing, *A.b.*, is arteria basilaris; *S.r.*, sinus rectus; *N.oc.*, nervus oculomotorius; *L.qu.*, lamina quadrigemina; *C.m.*, corpus mamillaria; *M.i.*, massa intermedia; F., fornix; *J.*, infundibulum; *Ch.*, Chiasma opticum; *C.c.*, corpus callosum; *N.o.*, nervus opticus; *H.*, hypophysis; *D.s.*, dorsum sellae; *S.sph.*, sinus sphenoidalis; *D.*, dura mater; *O.t.*, ostium tubae.

ANATOMY

The pituitary gland (hypophysis) lies in a groove of the roof of the sphenoidal sinus, the sella turcica. It is limited in front by the tubercle of the sella (olivary eminence), posteriorly by the dorsum sellae. The optic chiasma lies up to 1.5 cm.

23. Hirsch, O.: Genitalstörungen bei Hypophysentumoren, Wien. klin. Wchnschr. **44**:932, 1931.

24. Dandy, W. E.: Ventriculography Following the Injection of Air into the Cerebral Ventricules, Ann. Surg. **68**:5, 1918; Roentgenography of the Brain After the Injection of Air into the Spinal Canal, ibid. **70**:397, 1919.

25. Sgalitzer, M.: Use of Ascending Lipiodol, Fortschr. Geb. Röntgenstrahlen **36**:1100, 1927.

26. Schüller, A.: Contrast Filling of the Basilar Cisternae with Heavy Iodized Oil, Fortschr. Geb. Röntgenstrahlen **54**:251, 1936; Radiography of the Subarachnoid Cisternae at the Base of the Brain, Brit. J. Radiol. **13**:127, 1940.

27. Egas Moniz, A. C.: L'encéphalographie artérielle: Son importance dans la localisation des tumeurs cérébrales, Rev. neurol. **2**:72, 1927.

above the chiasmatic sulcus, the average distance being 0.75 cm. (Fig. 1). The space between the sulcus and the chiasm is filled by cerebrospinal fluid (chiasmatic cistern—Zander,[28] Schaeffer [29]). A tumor of the pituitary gland has to grow in most cases 0.5 cm. in order to reach the chiasm and has to grow higher up in order to flatten the chiasma (Hirsch and Kotrnetz [30]). One can see in the isolated specimen of a pituitary gland that it is covered by dura only. The pia arachnoid covers the stalk of the pituitary body, but does not reach beyond the diaphragm (Erdheim [14] and Wislocki [31]). The tumors developing from the anterior lobe of the gland—the posterior lobe is not a site of primary tumors—are therefore extradural. That explains why the mortality even in the preantibiotic era, was as low as 5%.

HISTOLOGY

The pituitary gland is constituted of three types of cells, named according to their affinity for stain: eosinophile, basophile (having affinity for hematoxylin), and chromophobe cells (not stainable by either eosin or hematoxylin). In an adult pituitary gland the eosinophiles are most numerous, the chromophobes least numerous. There is no agreement in this respect among the authors (Erdheim,[14] Rasmussen [32]). Though the chromophobe cells are least numerous, the incidence of chromophobe tumors is greatest; next in frequency are the eosinophile tumors, the ratio being about 3:1. The fourth cell type—frequent but not constant— is the squamous epithelial cells. These are remnants of the embryonal development from

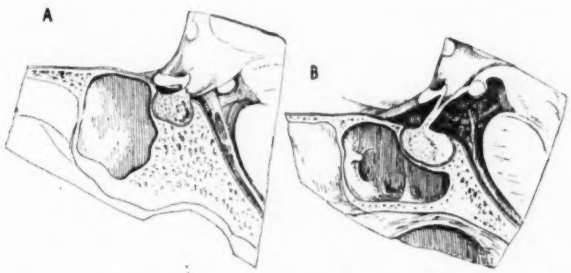

Fig. 2.—The extremes in the anatomical relation of the optic chiasma to the sulcus chiasmatis are illustrated. *A*, the chiasma touching the sulcus (so-called prefixed chiasma); *B*, the chiasma 1.4 cm. above the sulcus (postprefixed chiasma).[30]

28. Zander, R.: Lage und Dimensionen des Chiasma, Deutsche med. Wchnschr. (Supp.) **23**:13, 1879.

29. Schaeffer, J. P.: Some Points in the Regional Anatomy of the Optic Pathway, with Especial Reference to Tumors of the Hypophysis Cerebri and Resulting Ocular Changes, Anat. Rec. **28**:243, 1924.

30. Hirsch, O., and Kotrnetz, H.: Über die topographischen Beziehungen des Chiasma opticum zur Hypophyse und ihre Bedeutung für die Chirurgie des Hirnanhanges, Arch. klin. Chir. **168**:85, 1931.

31. Wislocki, G. B.: Meningeal Relations of the Hypophysis Cerebri: Relation in Adult Mammals, Am. J. Anat. **61**:95, 1937.

32. Rasmussen, A. T.: Proportions of the Various Subdivisions of the Normal Adult Human Hypophysis Cerebri and Relative Number of Different Types of Cells in Pars Distalis, with Biometric Evaluation of Age and Sex Differences and Special Consideration of Basophilic Invasion into Infundibular Process, A. Res. Nerv. & Ment. Dis., Proc. **17**:118, 1938.

the oral cavity. They occasionally give rise to hypophyseal-duct tumors (Erdheim,[14] or craniopharyngiomas (Cushing).[19] Cells appearing during pregnancy (Erdheim and Stumme)[33] or after thyroidectomy can be disregarded as far as tumor formation is concerned.

CLASSIFICATION OF PITUITARY TUMORS

The pituitary tumors can be classified as follows:

1. Intrasellar tumors, which make up about 15% of all 383 pituitary tumors, operated on by me until 1951, are almost synonymous with eosinophile tumors. These tumors grow exclusively toward the sphenoidal cavity and are therefore acces-

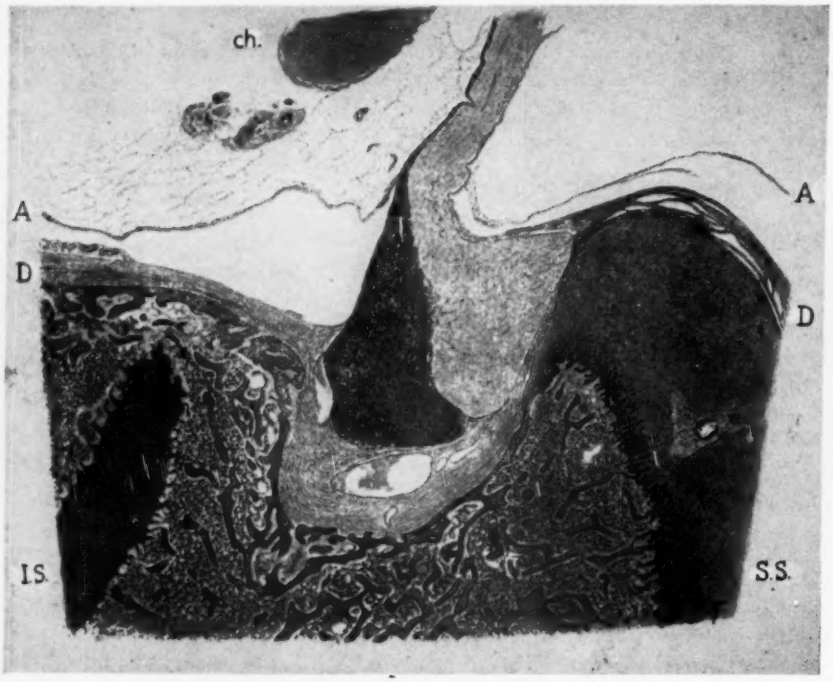

Fig. 3.—Section through the base of the skull of a fetus with the pituitary body: *D*, dura and its continuation forming the diaphragm of the sella. The arachnoid does not extend below the diaphragm. *I.S.*, intersphenoidal synchrondrosis; *S.S.*, spheno-occipital synchrondrosis; *ch.*, chiasma (Hirsch).

sible to the transsphenoidal operation. They do not interfere with the optic nerve or chiasma, and are usually small, no larger than a cherry. But they produce a secretion which causes acromegaly and often unbearable headache.

2. Craniosellar tumors are the most frequent of pituitary tumors, comprising about 60%, and are usually of the chromophobe type. They grow progressively in both directions toward the cranial cavity, and toward the sphenoidal sinus. At times

33. Erdheim, J., and Stumme, E.: Über die Schwangerschaftsveränderung der Hypophyse, Beitr. path. Anat. u. allg. Path. **46**:1, 1909.

the cranial extension prevails, at times the sphenoidal extension. They encroach upon the chiasma and produce progressive visual disturbances. They destroy the normal gland and lead to loss of function of the reproductive organs.

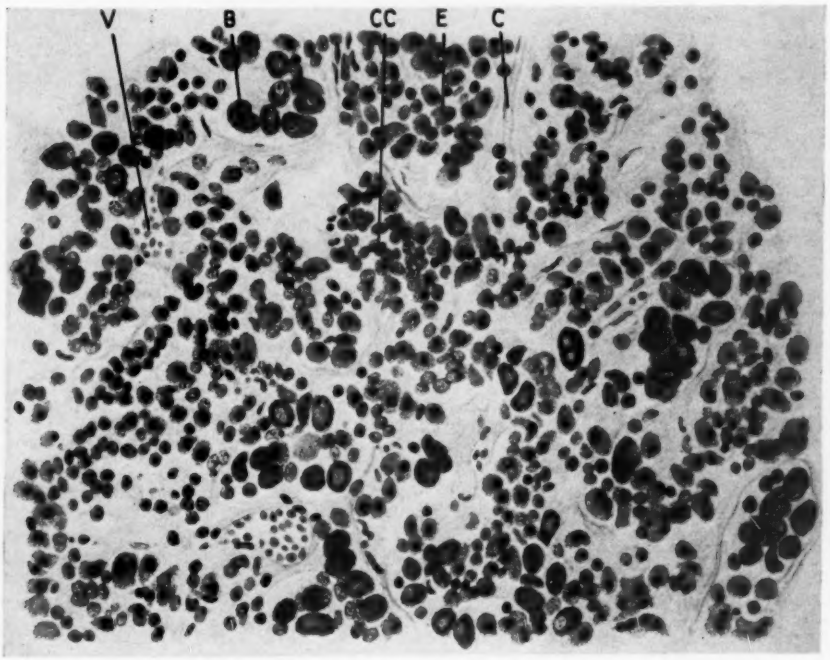

Fig. 4.—Microscopic picture of the anterior lobe of the pituitary gland. *E. C.*, eosinophile cells; *B*, basophile cells; *C. C.*, chromophobe cells; *V.*, vessel.[15]

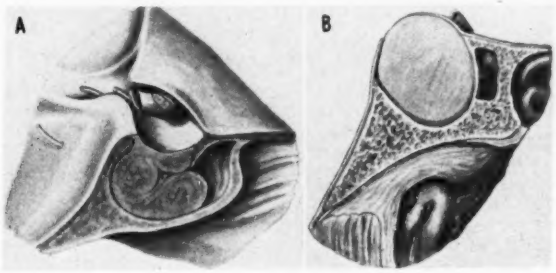

Fig. 5.—*A*, intrasellar tumor. The diaphragm is concave; the tumor has grown into the sphenoidal cavity. Optic nerve and ophthalmic artery are above the level of the tumor.[2] *B*, a benign craniosellar tumor grown toward both the sphenoidal and the cranial cavity producing bulging of the diaphragm (Institute of Pathology, Vienna).

3. Cystic and semicystic tumors constitute about 35% of my series of pituitary tumors. They represent cystic degeneration of chromophobe tumors, or craniopharyngiomas. They are especially amenable to the transsphenoidal operation.

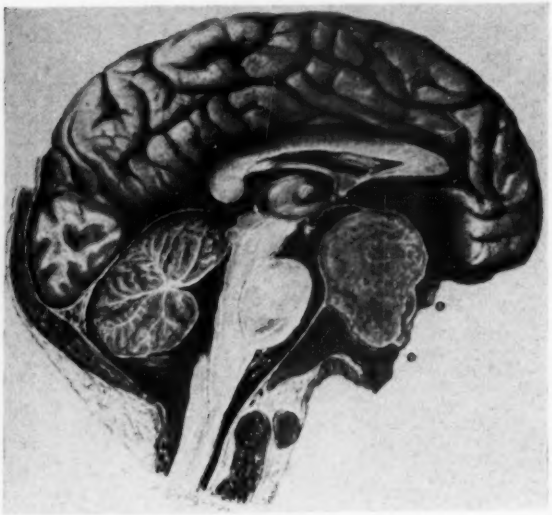

Fig. 6.—Specimen of a large craniosellar tumor with cerebral extension in a 57-year-old woman (operated on by me April, 1922) who died in October, 1928, in diabetic coma. Advanced visual disturbances occurred. The operative defect leading to the tumor is visible.

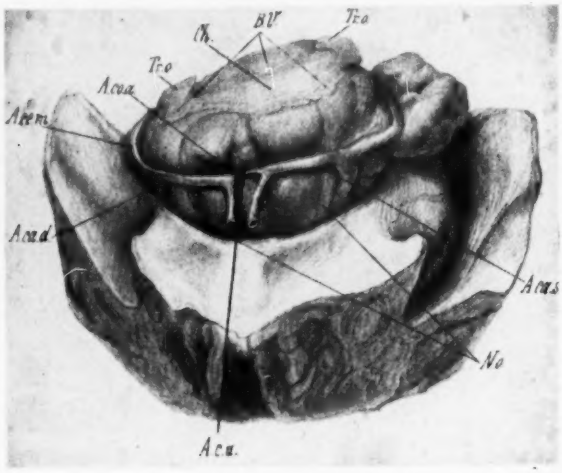

Fig. 7.—Malignant pituitary tumor with a cerebral extension. Chiasma (*Ch.*) is flattened. Nervi optici (*N.o.*) are strangulated by the circle of Willis. *B.V.*, floor of the third ventricle; *Tr.o.*, tractus opticus; *A. c. a.*, arteria communicans anterior; *A. ca. d.* and *A. ca. s.*, arteria carotis dextra and sinistra; *A. ce. m.*, arteria cerebri media (Hirsch [20]).

I did not mention basophile adenomas, which are an occasional cause of the well-known picture of Cushing's disease.[34] They are rare, small, and hardly subject to operation. They fall under Group 1.

All these tumors develop extradurally or are extradurally situated. Pituitary tumors which grow laterally (parasellar tumors) or lie above the diaphragm are located entirely within the cranial cavity and are not in the realm of transsphenoidal surgery.

DIAGNOSIS OF THE PITUITARY TUMORS

The diagnosis of the pituitary tumors is based on the clinical (endocrinological), ophthalmological, and x-ray findings.

Endocrinological Findings.—Among the clinical manifestations are (1) acromegaly, (2) Fröhlich's syndrome with amenorrhea (loss of potency) and visual disturbances, and a third type (ophthalmic) in which there is neither acromegaly nor Fröhlich's syndrome, whose chief symptom is visual disturbances. The latter are preceded or accompanied by amenorrhea or impotence. The pale complexion, the thin growth of hair, sleepiness, and indifference are conspicuous (Hirsch).[20]

Fig. 8.—Cystic tumor of the pituitary body (Hirsch).

Eye Symptoms.—Pituitary tumors are known to produce bitemporal hemianopsia. The latter represents a very advanced stage of damage to the optic pathways. Beginning encroachment upon the chiasma manifests itself by slight temporal restriction of the field for colors or by central scotoma. These field defects may progress with the growing tumor to the point of loss of a temporal quadrant on one or both sides, and later to temporal or bitemporal hemianopsia; finally, one quadrant on each side may remain from the visual field, or blindness of one or both eyes may result. The analogous field restrictions pertain also to homonymous hemianopsia.

34. Cushing, H.: Basophile Adenomas of the Pituitary Body and Their Clinical Manifestations (Pituitary Basophilism), Bull. Johns Hopkins Hosp. **50**:137, 1932.

I have found hemianopsia, i. e., restriction or loss of the temporal field of one eye (the other being normal or blind), and bitemporal hemianopsia in 77% of the cases of pituitary tumors without acromegaly.[35] If I include bitemporal hemianopsia for colors with normal fields for white, the percentage rises to 84.

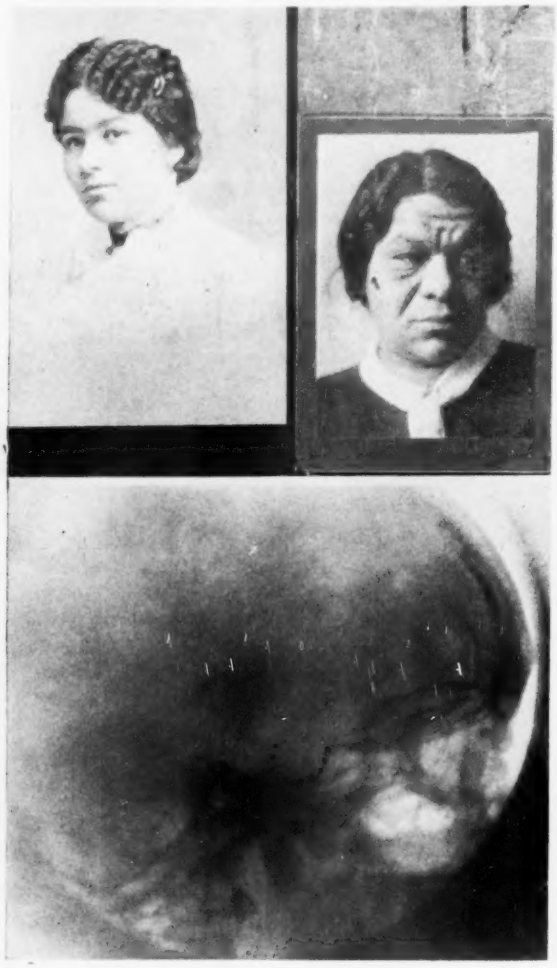

Fig. 9.—Acromegaly. Patient (left) before onset of illness and (right) at the time of the operation. Roentgenogram of the enlarged sella.

This percentage differs from that stated by Walker and Cushing,[36] who found bitemporal hemianopsia in 31.7% of their cases. This percentage pertains to

35. Hirsch, O.: Über Augensymptome bei Hypophysentumoren und ähnlichen Krankheitsbildern, Ztschr. Augenh. **45**:294, 1921.

36. Walker, C. B., and Cushing, H.: Distortions of the Visual Fields in Cases of Brain Tumors, Arch. Ophth. **47**:119, 1918.

bitemporal hemianopsia only, whereas figures given by me pertain to temporal and bitemporal hemianopsia. It is true that cases with temporal hemianopsia in one eye —the other eye being normal or blind—cannot be used in determining the percentage of bitemporal hemianopsia. Unilateral temporal hemianopsia can be a precursor or remnant of a homonymous hemianopsia. It was possible in most of my cases of

Fig. 10.—This 49-year-old man represents the ophthalmic type. His main complaint was of visual disturbances; there was no acromegaly and no conspicuous adiposity. There was absence of pubic and axillary hair.

unilateral hemianopsia to ascertain by the case history whether or not the blindness started with restriction of the temporal side.

The homonymous hemianopsia occurred in 7% of my cases. Unilateral nasal hemianopsia, i. e., loss of the nasal visual field of one eye, occurred in 4.4% of my cases and can be looked upon as a precursor of homonymous hemianopsia. On this basis the incidence of homonymous hemianopsia in my cases would amount to 11.4%. I found central scotomata mostly at the onset of the illness. At that time

they were erroneously taken for neuritis retrobulbaris and multiple sclerosis. Uncharacteristic visual fields were found in 7%; they were exclusively cases of papilledema.

Primary atrophy of the optic disks or pale disks was observed in 89% of my cases, papilledema in 8.9%, and neuritis optica in none, but postneuritic atrophy in 4.4%. Papilledema and neuritis were found altogether in 13.3% of my cases. (The total of 102.3% is due to the fact that in three patients the eyes had different findings or showed different findings at earlier stages of the illness.) The presence of papilledema or neuritis is a symptom which a priori warns against the diagnosis of pituitary tumor, in my opinion.[35] In such cases pneumoencephalography should be performed before one assumes the diagnosis of a pituitary tumor.

The visual findings in acromegaly do not differ from those in cases without acromegaly, but about 50% of acromegalics are spared visual disturbances, even if the acromegaly progresses.[35] This observation can be explained by the fact that about 50% of acromegalies are caused by small stationary tumors, developing

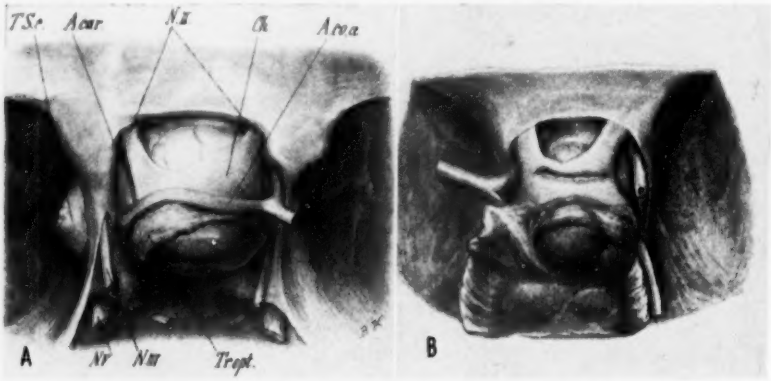

Fig. 11.—*A*, circle of Willis (anterior communicating artery) cutting into the chiasma, which, lying on the top of the tumor, presents accordingly, a convex contour. Its central prominent portion, containing the crossed fibers, is damaged first; later the sloping, lateral parts of the chiasma with its uncrossed fibers. *B*, Sulcus in the chiasma due to strangulation of the anterior communicating artery of the circle of Willis (Hirsch).[35]

beneath the diaphragm and not encroaching upon the optic pathways. The constant secretion of these tumors, not so much their growth, is responsible for the progress of acromegaly.

Bitemporal hemianopsia was systematically used for localization of a tumor compressing the chiasm only after the knowledge of semidecussation of the optic nerve had been established (von Gudden,[16] 1874). Homonymous hemianopsia was used much earlier for localization of a lesion in the visual centers. The mechanism of the strangulation of the optic chiasma by the circle of Willis was described in 1892 by W. Türck,[37] the founder of otolaryngology, at that time a neurologist.

The designation "pale disk" or "atrophy of the disk" is not identical with real atrophy of the optic fibers. The microscopical examination of such optic nerves

37. Türck, L.: Über Compression und Ursprung des Sehnnerven, Ztschr. Gesellsch. Ärzte Wien. **8**:299, 1852.

by Walker and Cushing (1915) [38] revealed that the amount of optic fibers preserved is far beyond that expected, judging from the color of the disk. Therefore pale disks are not always a sign of an irreversible loss of function and may not lead to a hopeless prognosis.[35] A surprisingly far-reaching recovery of the acuity of vision and the fields of vision has been observed.[35]

Besides the mentioned disturbances of the acuity of vision and the visual fields and the changes in the disk, one can find in pituitary tumors partial or complete paralysis of the external ocular muscles—mostly those supplied by the third, and infrequently those by the fourth, nerve—nystagmus, hypersensitivity of the vestibular apparatus, and, rather often, disturbances of the olfactory nerve. In one of my series I found more than 11% of such cases.[35]

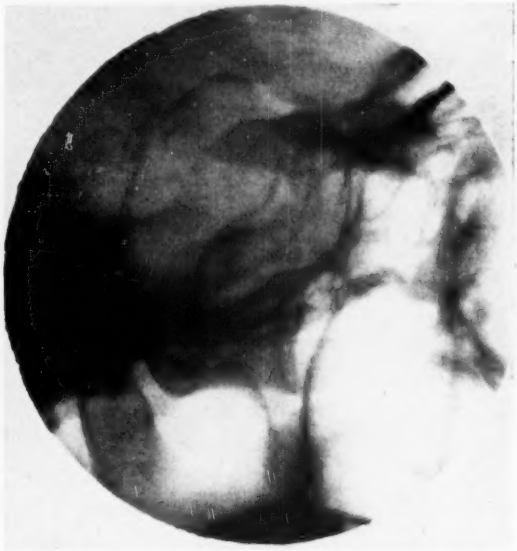

Fig. 12.—Convex contour above the sella corresponding to the calcified wall of a cystic tumor (craniopharyngioma).

X-Ray Findings.—X-ray findings are of different degrees. They may consist of the enlargement of the sella only, or of the decalcification of the floor of the sella. There may be reclination, deformity, or destruction of the dorsum sellae, and even complete destruction of the sella. Calcifications in the region of the sella are indicative of craniopharyngiomas (Algyogyi,[39] Luger [40]).

Genital Disturbances.—At this time I should mention the importance of the genital disturbances for the diagnosis. In 93.7% of all cases of chromophobe tumors

38. Walker, C. B., and Cushing, H.: Studies of Optic Nerve Atrophy in Association with Chiasmal Lesions, Arch. Ophth. **14**:407, 1916.

39. Algyogyi, H.: Ein Fall von radiologisch nachgewiesenem Tumor der Hirnbasis, Fortschr. Geb. Röntgenstrahlen **14**:257, 1909.

40. Luger, A.: Zur Kenntnis der im Röntgenbild sichtbaren Hirntumoren mit besonderer Berücksichtigung der Hypophysengangsgeschwülste, Fortschr. Geb. Röntgenstrahlen **21**:605, 1914.

decrease or loss of function of the reproductive organs has occurred. In cases of acromegaly genital disturbances were present in only 57%; I believe that genital disturbances were absent in the remaining 43% of acromegalics because of the small size of the tumor, which allows enough normal tissue to remain.[23]

1. 45-year-old man (Chiari - Hirsch [20]) acromegaly — Anterior lobe partly destroyed by an eosinophilic tumor; spermatogenesis with spermatozoa
2. 53-year-old man (Cushing-Davidoff *) acromegaly — Hypophysis of normal size, a small adenoma within the anterior lobe which is otherwise normal; spermatogenesis imperfecta
3. 21-year-old man (Chiari - Hirsch [20]) diabetes insipidus — Suprasellar cystic tumor extending into the anterior lobe, destroying it except for a thin layer; almost no spermatogenesis
4. 35-year-old woman (Hirsch [20]) ophthalmic type — Anterior lobe destroyed except for a thin layer; menopause for 4 years (ovaries not examined microscopically)
5. 40-year-old man (Cushing-Davidoff *) acromegaly — Large eosinophilic tumor with complete destruction of the anterior lobe; no spermatogenesis
6. 46-year-old man (Hirsch [23]-Hamperl) ophthalmic type — Superodiaphragmatic cystic tumor; normal pituitary gland; spermatogenesis
7. 53-year-old man (Erdheim †) acromegaly — Infrasellar eosinophilic tumor; pituitary gland normal; spermatogenesis

Fig. 13.—Genital disturbances in patients with pituitary tumors (autopsied patients). Dotted areas correspond to eosinophilic tumors; gray areas to remnants of anterior lobe. Behind them is the posterior lobe. * Cushing, H., and Davidoff, L. M.: Studies in Acromegaly: Pathological Findings in Four Autopsied Cases of Acromegaly with the Discussion of Their Significance, Monograph 22, New York, Rockefeller Institute for Medical Research, 1927. †Erdheim, J.: Über einen Hypophysentumor von ungewöhnlichen Sitz, Beitr. path. Anat. u. allg. Path. **46**:233, 1909.

DIFFERENTIAL DIAGNOSIS

The coincidence of the appearance of an enlarged sella in a roentgenogram, of bitemporal hemianopsia in its different stages, and of the clinical symptoms mentioned is in the prevalent majority of cases sufficient for the diagnosis of a pituitary tumor. But there are cases in which the diagnosis is difficult, for instance, those of an enlarged sella without visual disturbances, or cases in which one eye is blind, and the other normal. Also, in treating children or women over 50, or women who have undergone hysterectomies with absence of reproductive-organ function, even an experienced physician can be faced with difficulties. Contrast filling by air

(Fig. 14.*A*) or by iodized oil (lipiodol®) (Sgalitzer,[25] Schüller [26]) and arteriography (Egas Moniz [27]) can be of great help.

Each of the diagnostic resources—enlarged sella, bitemporal hemianopsia, and even a convex contour above the sella in contrast filling—is by itself not entirely reliable. For instance, an enlarged sella can be found in hydrocephalus.[28] Bitempo-

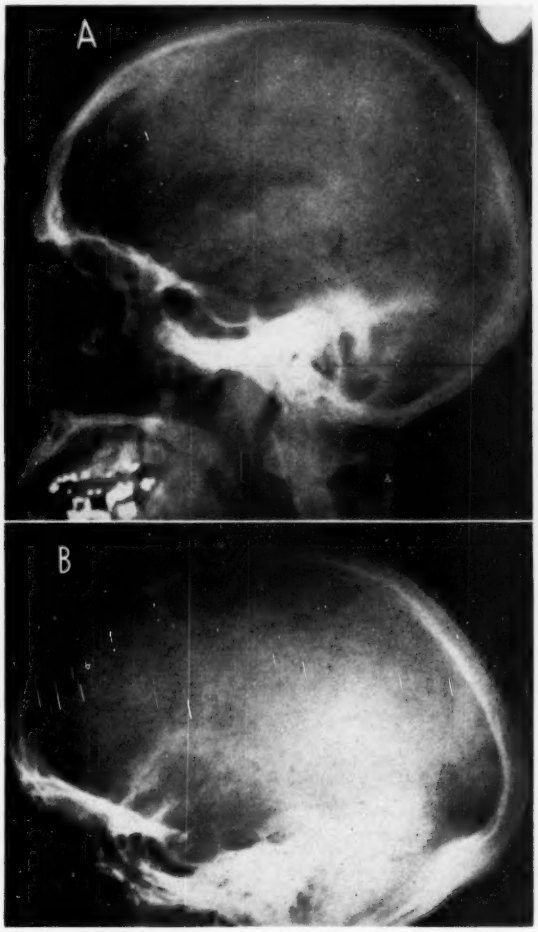

Fig. 14.—*A*, air encephalogram by Dr. H. Hamlin revealing an egg-shaped tumor which in the operation (Hirsch), July 18, 1950, proved to be cystic. *B*, Roentgenogram of a 60-year-old woman with failing vision, enlarged sella, and calcification within the region of the sella.

ral defects or hemianopsia is a regular finding in meningiomas of the sellar region without enlargement of the sella (Cushing [19]). It can be found also in gliomas of the chiasma or of adjacent structures with enlarged sella, as I mentioned in an earlier article.[41] Calcifications which are characteristic of craniopharyngiomas can be

41. Hirsch, O.: Gliom in der Gegend der Lamina terminalis unter dem Bilde eines Hypophysentumors, Jahrb. Psychiat. u. Neurol. **48**:208, 1932.

found in aneurysms, chondromas, osteomas, and sclerotic carotid arteries. Knowing the value of these symptoms, one can make the diagnosis of a pituitary tumor and avoid errors with great certainty, but exceptions exist.

Since we learn more by errors than by diagnosis which proves correct, I should like to present brief histories of two cases in which the diagnosis of a pituitary tumor was missed.

A 30-year-old man was admitted in 1932 to the eye department of the University of Vienna because of failing vision. Bitemporal hemianopsia, diminished acuity of vision, 3/60 and 6/60, and genuine atrophy of the disk were found. Normal distribution of hair and absence of genital disturbances warned against the diagnosis of a pituitary tumor. But contrast filling showed a convex contour above the sella. A transsphenoidal operation proved the diagnosis to be an error. A glioma of the lamina terminalis reaching into the sella was found post mortem.[41]

In this case the diagnosis of a pituitary tumor was assumed, but proved to be incorrect.

In the case which follows, a pituitary tumor was present, but the diagnosis was not sufficiently certain to risk a craniotomy.

A 60-year-old woman was treated one year previously (1950) at the Boston City Hospital because of failing vision. The sella in the roentgenogram appeared enlarged. Calcification in the region of the sella and a bruit over the orbit were present. Arteriography was inconclusive, and the risk of craniotomy seemed excessive. Vision continued to fail. A transsphenoidal operation was performed eight months later by me. Since aspiration by needle did not show any fluid or blood, the dura was incised, and a chromophobe tumor was encountered. The patient regained vision.

TREATMENT

There are four methods of treating pituitary tumors: medical, x-ray, and transfrontal and transsphenoidal operations. Each method has its limitations.

Medical Treatment.—Medical therapy is limited to that type of acromegaly which produces endocrine symptoms only, i. e., progressive acromegaly with headache and with paresthesia or numbness, or even aching, of the fingers. The response to estrogen therapy is often favorable (Albright and associates [42]).

X-Ray Treatment.—The results for three series are tabulated.

	Dyke and Hare [43] Follow-up of Eight Years	Sosman [44]	Davidoff and Feiring [45]
Chromophobe tumors			
Improved	26.4%	50%	.....
Favorably affected	.....	....	42.4%
Became worse	47.3%	....	12%
Eosinophile tumors			
Improved	40%	90%	68%
Not improved	.....	....	26.6%
Worse	24%	....	.....

42. Albright, F.: Reifenstein, E. C., Jr., and Kinsell, L. W.: Effect of Estrogen Therapy in Acromegaly with Observations on the Use of the Serum Phosphorus Level as an Index of Pituitary Growth Hormone Activity, J. Clin. Invest. **25**:932, 1946.

43. Dyke, C. G., and Hare, C. C.: Roentgen Therapy of Pituitary Tumors: Report of 63 Cases, A. Res. Nerv. & Ment. Dis., Proc. (1936) **17**:651, 1938.

44. Sosman, M. C.: Irradiation in the Treatment of Pituitary Adenomas, Proceedings of the Interstate Post-Graduate Medical Assembly of North America, St. Louis, Oct. 18, 1937.

45. Davidoff, L. M., and Feiring, E. H.: Surgical Treatment of Tumors of the Pituitary Body, Am. J. Surg. **75**:99, 1948.

X-ray treatment was successfully started by Béclère,[46] Paris, in 1909, and by his pupil Gramegna,[47] Turin, in 1909. X-ray treatment of the pituitary tumors is assumed to be a procedure which does not involve any risk or undesired effects except epilation. That is generally true, though incidents of cerebral disturbances (Sosman [44] and one personal observation) among the immediate sequelae and the development of cataract one to seven years later have been observed after irradiation (Thiel [48]).

Operative Treatment.—The transfrontal method is difficult in instances of a prefixed chiasma, manifesting itself by central scotomata. Such a chiasma impedes the access to the tumor. Cushing [19] had to resort several times to the dissection of the chiasm; other neurosurgeons prefer to cut one optic nerve or to resect a part, at times all, of a frontal lobe, in order to gain access to the tumor (Dandy [49]).

In persons over 60 with shrunken convolutions and dura strongly adherent to the bone, the transfrontal operation may result in mental disorders (Cairns [50]). The mortality rate of the transfrontal method, according to Grant,[51] is 11.2%, excluding craniopharyngiomas, which have a mortality rate of 35%.

The kind of treatment about which I can speak from my own experience is the trans-sphenoidal-septal method.

HISTORY OF TRANSSPHENOIDAL OPERATION

The first attempts to remove pituitary tumors utilized the cranial route: through the middle fossa by Horsley [52] in 1904, and through the anterior fossa by Krause [53] in 1905. It was the high mortality and the technical difficulty which directed surgeons, not the rhinologists, to the transsphenoidal method. This technique was first suggested by Schloffer [54] in 1906 and performed by him [55] in 1907. He used the dislocation of the external nose by a lateral incision. This operation became popular among surgeons in the first decade after its publication. More than 100 operations of this kind were performed at that time by various surgeons. By this method

46. Béclère, M.: Le traitement médical des tumeurs hypophysaires, du gigantisme et de l'acromégalie par la radiothérapie, Bull. mém. Soc. méd. hôp. Paris, Ser. 3 **26**:274, 1909.

47. Gramegna, A.: Un cas d'acromégalie traité par la radiothérapie, Rev. neurol. **17**:15, 1909.

48. Thiel, R.: Zur Klinik und Therapie der Hypophysengeschwülste, Klin. Monatsbl. Augenh. **90**:581-597, 1933.

49. Dandy, W. E.: Surgery of the Brain, Hagerstown, Md., W. F. Prior Company, 1945, pp. 554, 562, 570.

50. Cairns, H.: Ultimate Results of Operations for Intracranial Tumors: Study of Series of Cases After 9-Year Interval, Yale J. Biol. & Med. **8**:421, 1936.

51. Grant, F. C.: Surgical Experience with Tumors of the Pituitary Gland, J. A. M. A. **136**:668 (March 6) 1948.

52. Horsley, V.: Address in Surgery: On the Technic of Operations on the Central Nervous System, Brit. M. J. **2**:411, 1906; cited by Verga, G., in Patologia chirurgicale delle l'ipofisi, Pavia, 1911.

53. Krause, F.: Hirnchirurgie (Freilegung der Hypophyse), Deutsche Klin. **8**:1004, 1905.

54. Schloffer, H.: Zur Frage der Operationen an der Hypophyse, Beitr. klin. Chir. **50**:767, 1906.

55. Schloffer, H.: Erfolgreiche Operation eines Hypophysentumors auf nasalem Wege, Wien. klin. Wchnschr. **20**:621, 1075, 1907.

von Eiselsberg [56] operated on the patient in the original case of Fröhlich's syndrome, in 1907, and Hochenegg [57] was the first to operate on a patient with acromegaly, in 1908. The mortality rate of this method was 25%.[56] Other surgeons used different ways of dislocating the external nose in their attempt to reach the sphenoidal sinus: Dislocation from above downward was used by Proust [58] in 1908; from below upward by external incision, by Kanavel (1909) [59] and Kanavel and Grinker (1910),[60] and by sublabial incision, by Halstead (1910).[61] I suggested in 1909 and performed successfully in 1910, with local anesthesia,[62] a transsphenoidal operation endonasally. This first operation was based on the opening of one sphenoidal cavity by the ethmoidal route (Hajek).[63]

Because the access was unilateral, I worked out and used in all subsequent cases a septal access to both sphenoidal sinuses, based on submucous resection of the septum [64] (1910, 1912). Cushing combined the sublabial incision of Halstead with my septal method, calling it his "favored method . . . to all intents and purposes, similar to the endonasal method which Hirsch has recently described and which he has successfully practised in a number of cases" (Cushing [65]).

The rhinologists very soon became interested in the surgery of pituitary tumors and devised, according to their liking, various methods: the paranasal method (Chiari,[66] 1912), using the external ethmoidectomy; the buccal method, through the maxillary sinus (Fein,[67] 1910; Denker [68]), and the palatinal method (Preysing,[69]

56. von Eiselsberg, A., and von Frankl-Hochwart, L.: Über operative Freilegung der Tumoren in der Hypophysengegend, Neurol. Zentralbl. **26**:994, 1907. von Eiselsberg, A.: My Experience About Operations upon the Hypophysis, Tr. Am. S. A. **28**:55, 1910; Über den Endausgang und die Obduktion meiner ersten operierten Fälle von Hypophysentumor, Beitr. path. anat. **71**:619, 1922-1923; Die Hypophyse, Vienna, Urban & Schwarzenberg, 1930, pp. 34-37.

57. Hochenegg, J.: Zur Therapie von Hypophysentumoren, Deutsche Ztschr. Chir. **100**:317, 1909.

58. Proust, R.: Chirurgie de l'hypophyse, J. chir. **1**:668, 1908; Rev. neurol., 1909, p. 228.

59. Kanavel, A. B.: Removal of Tumors of the Pituitary Body by an Infranasal Route, J. A. M. A. **53**:1704 (Nov. 20) 1909.

60. Kanavel, A. B., and Grinker, J.: Removal of Tumors of the Pituitary Body, Surg., Gynec. & Obst. **10**:414, 1910.

61. Halstead, A. E.: Remarks on the Operative Treatment of Tumors of the Hypophysis: Report of Two Cases Operated on by an Oro-Nasal Method, Surg., Gynec. & Obst. **10**:494, 1910.

62. Hirsch, O.: (*a*) Eine neue Methode der endonasalen Operation von Hypophysentumoren, Wien. med. Wchnschr. **59**:636, 1909; (*b*) Offiz. Protokoll der K. K. Gesellsch. d. Ärzte in Wien, Wien. klin. Wchnschr. **23**:563, 1910.

63. Hajek, M.: Zur Diagnose und intranasalen chirurgischen Behandlung des hinteren Siebeinlabyrinthes, Arch. f. Laryng. **16**:105-143, 1904.

64. Hirsch, O.: Über Methoden der operativen Behandlung von Hypophysentumoren auf endonasalem Wege, Arch. Laryng. **24**:129, 1910; footnote 20.

65. Cushing, H.: The Pituitary and Its Disorders, Philadelphia, J. B. Lippincott Company, 1912, p. 300.

66. Chiari, O.: Über eine Modification der Schloffer'schen Operation von Tumoren der Hypophyse, Wien. klin. Wchnschr. **25**:5, 1912; Akromegalie infolge Hypophysentumors: Operation, ibid. **24**:955, 1911.

67. Fein, J.: Zur Operation der Hypophyse, Wien. klin. Wchnschr. **23**:1035, 1910.

68. Denker, A.: Drei Fälle operiert nach transmaxillarer Methode, Internat. Zentralbl. Laryng. **37**:225, 1921.

69. Preysing: Beiträge zur Operation der Hypophyse, Verhandl. deutsch. Laryng. **20**:51, 1913.

1913; Tiefenthal, 1920[70]). But none of these methods offers the advantages of the septal method, especially in regard to postoperative treatment and the treatment of recurrences. The septal method also permits repeated radium application, in order to prohibit regrowth of the tumor (Hirsch[71]).

MY ENDONASAL-SEPTAL METHOD

The operation consists of a submucous resection of the septum, which is carried out as far as the anterior walls of the sphenoidal sinus. When the flaps of the septum are spread by speculum to the width of both nasal cavities, a new cavity results which has no communication with the nasal cavities (except for the first incision) and is (essentially) abacterial. Removal of the rostrum of the sphenoid bone usually results in opening one or both sphenoidal sinuses. If this does not occur, the sphenoidal sinuses can be opened by infracting the anterior wall of each side of the

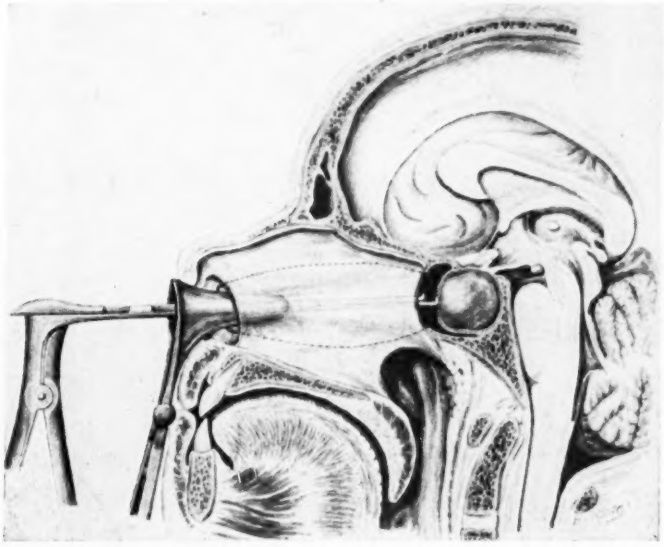

Fig. 15.—Hirsch's transsphenoidal-sella septum operation (stage of the removal of the bony shell of the sella).

rostrum. Through these openings the anterior walls of the sphenoidal sinus are removed by rongeurs.

If an intersphenoidal septum is present, it has to be removed. Then the enlarged sella becomes visible, and if it does not show a defect it has to be opened by chisel. From this opening the bony sella is removed by thinner rongeurs, and the bluish-colored periosteum becomes exposed. The tumor lies directly behind it. The exposure measures about 1.5 by 1.75 cm., more or less, depending upon the size of the enlarged sella. There is usually very little bleeding at this point. The field of operation can be kept free of blood by suction. After the periosteum has been exposed, I use another sterile set of instruments. The tumor is needled and aspirated. If fluid

70. Tiefenthal: Technik der Hypophysenoperation, Münchener med. Wchnschr. **67**:794, 1920.

71. Hirsch, O.: Über Radiumbehandlung der Hypophysentumoren, Arch. Laryng. **34**:133, 1919; Footnote 20.

is encountered, an incision of 1 to 2 mm. is made in order to allow the fluid to escape slowly. After the dripping of the fluid has stopped, the incision is enlarged and a flap is formed, which can be excised or—as I plan to do in the future—turned inside into the cavity of the cyst, in order to prevent the closure of the cyst.

Until now I have tried to prevent the closure of the cyst by keeping a tube in the cavity for several weeks, changing it if necessary. If the tumor is solid and soft, it can be removed to a greater or less extent by suction. If this procedure has insufficient effect, the tumor is curetted or pieces are removed with the Bovie snare.

One should refrain from thorough excochleation. It would only increase the chance of infection, without achieving a complete removal of the tumor. After careful and thorough suction of the operating field, the two flaps of the septum are put together. In this way the sphenoidal sinus is closed again by the mucous

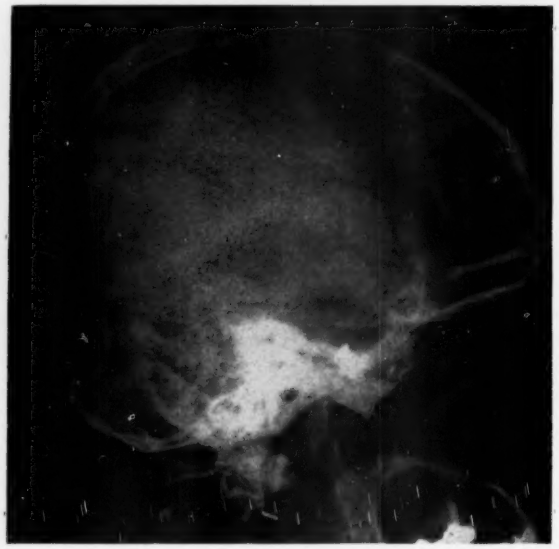

Fig. 16.—Metal tube introduced into the cystic tumor for the purpose of drainage.

membrane. In order to have an access for future radium treatment, I excise the membranous anterior sphenoidal walls and even the posterior part of the septum. To prevent bleeding oxidized cellulose U. S. P. (oxycel®) is applied. The operation is finished by placing petrolatum strips on both sides of the septum. They are removed 48 hours after the operation. Antibiotics are given on the day before the operation, and administration is continued for one week after the operation.

This operation can be done with the area under local anesthesia, but even if general anesthesia is used infiltration of the septum with procaine hydrochloride and epinephrine is advisable, because it restricts the bleeding and allows a constant good view.

This operation provides only partial removal of the tumor, but no other method exists by which a tumor of craniosellar extension can be removed radically without

endangering the patient's life. Since the craniosellar tumor causes visual disturbances, one can say that in a case with visual disturbances the tumor cannot be removed completely, either by the transsphenoidal or by the cranial method. Rare exceptions exist.

There was always the question even among rhinologists whether one can see enough of the field of operation and of the tumor. When the submucous resection of the septum is done, one separates the two flaps and keeps them apart by a nasal speculum, to the width of both nasal cavities. In this way one gets a field of opera-

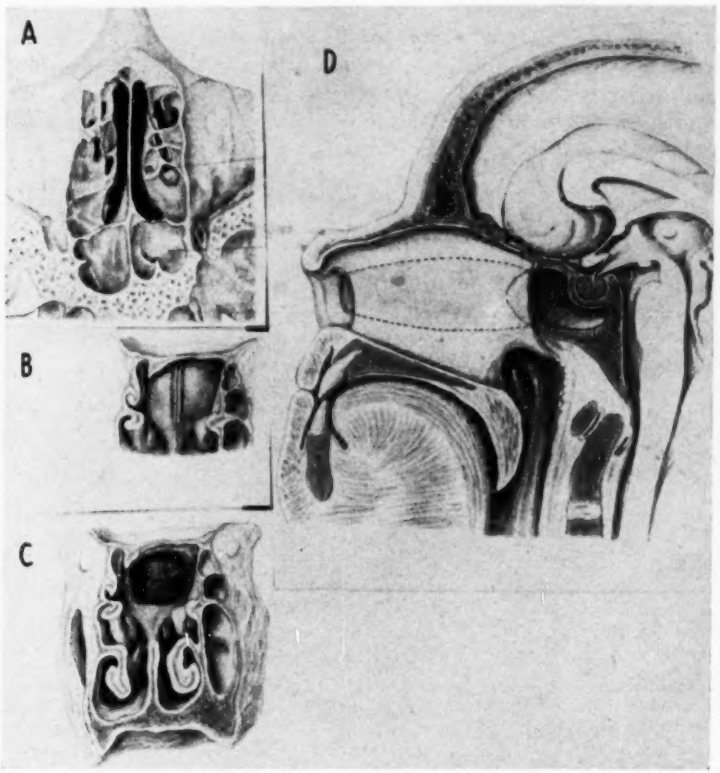

Fig. 17.—*A* shows that at the end of the operation, after the flaps of the septum were put together, the sphenoidal sinuses are closed by mucous membrane; *B* shows the incision; *C*, the excision of the anterior membranous walls of the sphenoidal cavities; *D*, the excision of the posterior part of the membranous septum (semicircular punctuated line).

tion as large as the two nasal cavities. Each point is visible in the reflected light. Until the periosteum of the sella (dura) is incised and the tumor curetted, the field of operation is as a rule dry and free of blood.

Chevalier Jackson assisted Lewis in a transsphenoidalseptal operation (sublabial incision) for a pituitary tumor.[72] This is his comment on the operation:

72. Jackson, C., in discussion on Lewis, F. O.: Pituitary Tumor: Operation by Transsphenoidal Route, Pennsylvania M. J. **47**:282, 1917.

It is remarkable what a large view can be obtained; what room there is in which to work. . . . The trans-sphenoidal route seems to me to be the one for a septal operation; it may not be the route of choice for the man who has not done a great many submucous septal operations. I should like to offer the evidence of this particular case as to the large field obtained, because I myself was surprised.

This evidence, though dating back to 1917, is still valid.

Lately there have been publications which describe the removal of the tumor in transfrontal operations as subtotal, or peeling out of the tumor. This method may be applied in very exceptional cases, but, as a rule, it is not possible.

To supplement transsphenoidal removal of the part of the pituitary tumor, radium is applied to the surface of the remnant of the tumor in order to destroy it or to prevent its regrowth. Radium treatment [71] is given after the healing has taken place, not before the sixth postoperative week. The radium carrier is oval, 1 to 1.5 cm. in diameter. It has a thin platinum wall toward the tumor; the other part

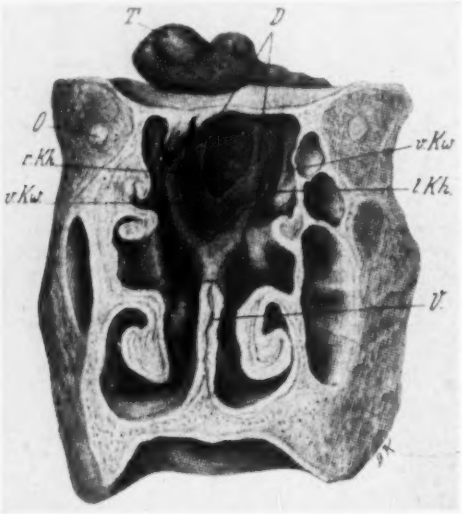

Fig. 18.—Specimen from the operative field of a patient operated on by the endonasal method by the author. The bony defect in the anterior walls of the sphenoid and the edges of the excised dura are visible.

is of thick platinum metal. It is adapted to a wire by a joint which allows the capsule to recline to 90 degrees only. The frequency and time of application depend upon the radium sensitivity of the tumor, whose shrinkage is manifested by increasing acuity and fields of vision.

The transsphenoidal method has the advantage that the tumor can be reached in the open sphenoidal cavities at any time when declining vision arouses the suspicion of recurrence. For instance, if the refilling of a cystic tumor is suspected, it can be punctured and reopened without much effort, with local anesthesia and two to three days' hospitalization. If a recurrence of a solid tumor occurs, additional radium application can be given at the office. In this way lasting results can be obtained. The treatment of cystic tumors particularly is so easy that no other method offers such an advantage.

Complications.—The complication most often encountered in the transsphenoidal method is meningitis. The previous mortality rate of 5% was mostly due to this complication. Today meningitis can be controlled by antibiotics. Meningitis develops apparently by infection of the tumor rest, not by direct infection of the sub-

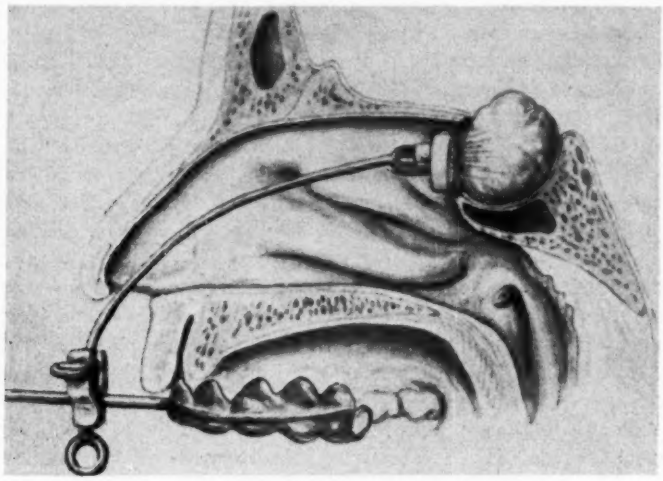

Fig. 19.—Radium applicator.[72]

Fig. 20.—Roentgenogram of the radium applicator in situ.[72]

arachnoidal space. Therefore, I have used suction of the operative field when suspicion of meningitis arose. I believe that by this method I prevented, in the pre-antibiotic era, the development of meningitis. At the present time I use both suction and antibiotics.

Another complication is the leakage of cerebrospinal fluid, which I have experienced twice in cases of eosinophile tumors without conspicuous visual disturbances.

In one case the leakage was combined with an aerocele. The field of operation in both these cases was limited by a moderately enlarged sella and small tumor. To close the leak by the endonasal method, I used a long flap from one side of the septum. This flap was turned by its raw surface into the sphenoidal cavity and pressed toward the leak and kept in this position by a petrolatum tampon. Meningitis developed in both cases in which I had used this method. The patients were cured in a few days by intrathecal injections of penicillin (25,000 units) and of dihydrostreptomycin (25 mg.). The leakage stopped after one week. I had the cooperation of the neurosurgeon, Dr. H. Hamlin, in the postoperative treatment in both cases, but particularly in the first, in which I used this method. This method promises to be the solution of endonasal treatment of cerebrospinal leakage. In chronic cases, the region of the leakage has to be refreshed by 25% silver nitrate solution in order that the flap may take. (This method was first described at the preliminary presentation of this paper.)

Operation in a case of acromegaly without visual disturbances and with a small sella led to the exposure of the cavernous sinus, owing to the extreme asymmetry of the two sphenoidal sinuses. Bleeding was easily stopped by gauze without any further complication. The gauze was removed after two weeks and the treatment completed.

One patient, who though improved immediately after the operation, had loss of vision on the following day. The patient regained some vision later but lost it again in a new cerebrovascular attack. A cranial operation, which was performed a half-year later, revealed that both internal carotid arteries were the seat of aneurysmal dilatations. This cannot be called a complication; rather it was a coincidence.[72a]

IMMEDIATE RESULTS

In the last six years I have operated on 37 patients in Boston. These patients presented various types of tumors, and I cite one or two cases of each type.

Intrasellar (Eosinophile) Tumors Without or With Slight Visual Disturbances, Causing Acromegaly.—I operated on 13 patients with tumors of this type, but not all of them had the excellent result obtained in the following case:

A. Z., a 37-year-old man with acromegaly, diabetes mellitus, polyphagia, and polyuria, was admitted to Rhode Island Hospital (Dr. H. Hamlin). The visual fields for white and color were normal. The fields of vision were becoming slightly restricted. He was treated with x-rays, with favorable, but insufficient, results. Therefore, an operation was suggested and carried out by me, in cooperation with Dr. Hamlin. The tumor was classified as adenoma by the pathologist, Dr. B. E. Clarke (Rock Island Hospital). Two years after the operation the patient was seen by Dr. Hamlin and me. The preoperative headache and back pain were gone. A few months after the operation he could stop the use of insulin. He wears gloves size 7½ instead of 8, as well as a smaller shoe size, and he has no numbness of the hands. Hair in the axilla and the genital region was scarce before the operation, but is plentiful now. The excessive sweating is gone; the skin of the face, which was hard, is smooth now. The tiredness, impatience, hunger, and nocturia are gone. Potency is restored.

Craniosellar Tumors.—I have operated on 15 patients with tumors of this type in the last six years. Fourteen had solid chromophobe tumors, among them two tumors microscopically diagnosed as malignant because they had infiltrated the bone marrow of the sphenoid. One patient of this group had a solid cranio-

72a. Brougham, M.; Housner, A. P., and Adams, R. D.: Acute Degenerative Changes in Adenomas of the Pituitary Body, with Special Reference to Pituitary Apoplexy, J. Neurosurg. 7:421, 1950.

pharyngioma. All these tumors caused visual disturbances. The intrasellar part of these tumors was first removed by operation. In this way a decompression was achieved. Afterward healing radium was applied, which further reduced the tumor rest, to differing extents, according to the sensitivity of the tumor. The tumor rest then may remain in the enlarged sella as a harmless foreign body.

Two case histories follow. The first is that of a patient with a chromophobe tumor with Fröhlich's syndrome, advanced visual disturbances, and paresis of external ocular muscles. The treatment resulted in practically full recovery from the ocular symptoms.

The second case is reported because of the advanced age of the patient.

B. D., a 24-year-old woman, ceased menstruating at 22 and became adipose. The sella was enlarged, and the right anterior and posterior clinoid processes were eroded. The transsphenoidal-septal operation was performed on Aug. 27, 1949. A chromophobe tumor was encountered. Radium was applied during the following three months. The right eye before the operation had temporal hemianopsia, with sparing of the macula and acuity of vision of 20/50. The left eye had a small nasal field with loss of the central area and the visual acuity was reduced to counting fingers at 3 ft. (0.9 meter), excentric. Besides that, the patient had double vision due to paresis of the oculomotor nerve. The disks were slightly pale. Now, one and a half years later, the visual acuity is 6/6 in each eye; the field of the left eye is normal, and the field of the right eye has extended temporally to 20 degrees. Double vision disappeared.

Among the patients with solid tumors in this group was a 75-year-old woman with steadily failing vision, whose age contraindicated transsphenoidal surgery. She could see movements of hands with the left eye; with the right eye she counted fingers at 6 ft. (1.8 meters). She had to be guided. I operated on her in September, 1946. After operation and radium application she regained useful vision. She is now 80, is doing well, and even does needle work.

Two patients, completely blind, came to the operation as their last hope. They remained blind in spite of operation and radium treatment.

Among the 15 solid tumors of craniosellar extension were two malignant tumors, one with distinct, the other with fugitive, acromegaly.

N. C., a 32-year-old woman with acromegaly, showed homonymous hemianopsia with visual acuity of 20/25 in the right eye and of 20/200 (without correction) in the left eye. The roentgenogram revealed complete destruction of the sella and of the sphenoid body. The tumor had grown into the nasal pharynx.

The patient was operated on on April 12, 1949. The tumor was partially removed and proved microscopically to be a malignant chromophobe neoplasm of the pituitary gland invading the bone marrow. Radium was applied after operation. The roentgenogram of February, 1951, showed that the sella had recalcified and that the pharyngeal extension of the tumor had shrunk. Visual acuity has improved to 20/13 in the right eye and to 20/70 in the left eye without correction and 20/25 with correction. The field of vision showed slight widening in the central area.

Cystic Tumors.—This type was presented by eight of the 37 patients operated on in the last six years. Three of them had cystic degenerated chromophobe tumors, five had craniopharyngiomas. The following is a brief history of a patient with a cystic degenerated chromophobe adenoma, who was treated first by tubes introduced into the cyst, in order to achieve permanent drainage, and later by radium, in order to prevent the growth of the solid part.

M. C., a 59-year-old woman noticed failing vision in October, 1947. A roentgenogram revealed an enlarged sella. The diagnosis of a pituitary tumor was made, and the patient was treated by x-rays, but the vision became worse. When I saw the patient a half year later, in April, 1948, a sharp bitemporal hemianopsia was found. Vision in the right eye was 20/100, in the left eye, 20/200. The disks were normal. Operation on April 7, 1948, revealed a cystic

degenerated chromophobe tumor. Within one week the patient regained normal acuity of vision and normal visual fields. Radium was applied during the following months. The patient had three recurrences of failing vision. Since the tumor was freely accessible in the open sphenoidal sinuses, the reopening of the cyst was a simple procedure. Vision returned each time in 24 hours.

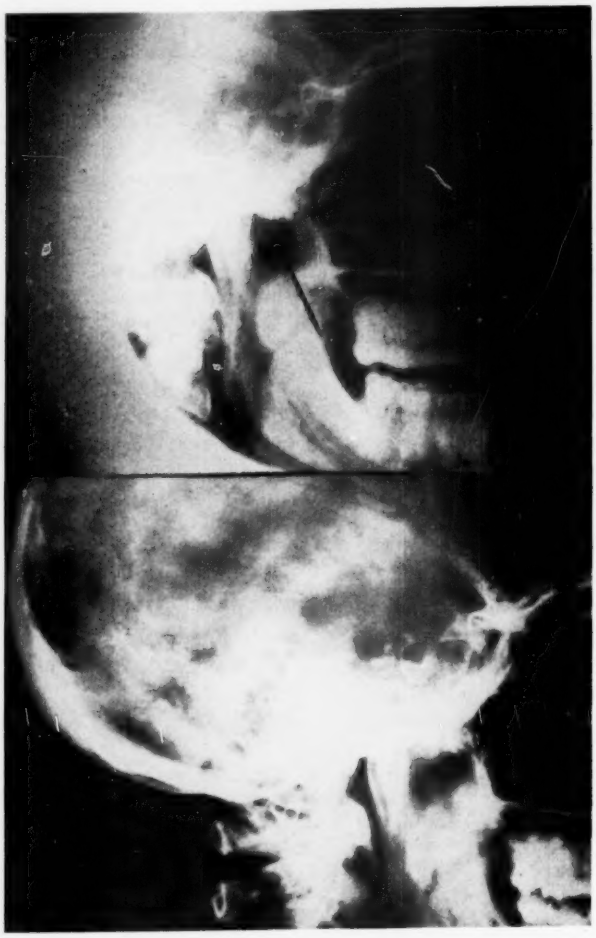

Fig. 21.—Roentgenogram of the 32-year-old woman with acromegaly, showing destruction of the sella and sphenoidal body and the tumor penetrating the nasopharynx (arrow). The roentgenogram below was taken nearly two years after the operation and radium treatment, and shows recalcification of the sella and shrunken pharyngeal extension.

Five of the cystic tumors were craniopharyngiomas. One of these occurred in an emaciated 19-year-old woman with Simmonds' disease (pituitary cachexia) who died three weeks after the operation, of a metabolic disorder. Brief histories of three patients follow, each history differing from the others: one patient with a

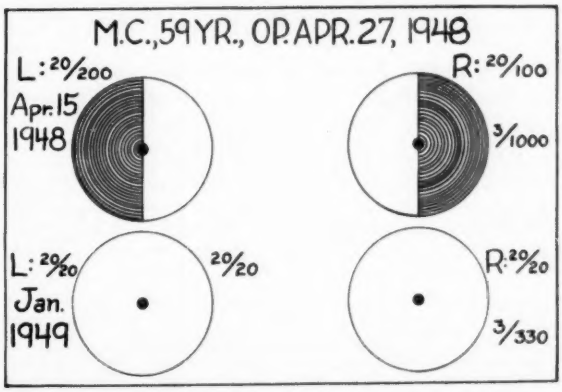

Fig. 22.—Fields of the patient M. C., a 59-year-old woman, before the operation and nine months later. After the recurrences the acuity of vision and the fields are the same today as in January, 1949.

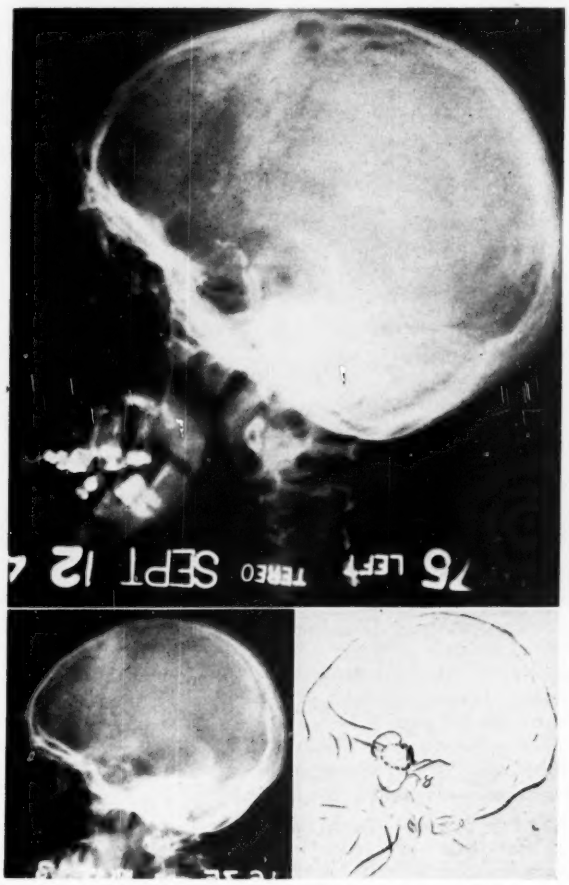

Fig. 23.—Roentgenograms of the patient R. B. before and after operation, showing shrinkage of the calcified tumors in the extent of the diagram.

large tumor without visual disturbances, one patient with homonymous hemianopsia, and the third patient, a 3½-year-old girl who belongs to the youngest group of patients successfully operated on for craniopharyngioma.

R. B., a 29-year-old man, suffered from attacks of headache, but had no visual disturbances. The contour of the tumor was visible in roentgenograms. At operation, in September, 1947, a cystic tumor with cheesy contents was encountered. An opening was established to let the contents escape. The cystic tumor measured 4.5 by 5 cm. The shrunken calcified shell, 2 to 3 cm. in diameter, remains as a harmless foreign body in the sella. The attacks of headache are gone.

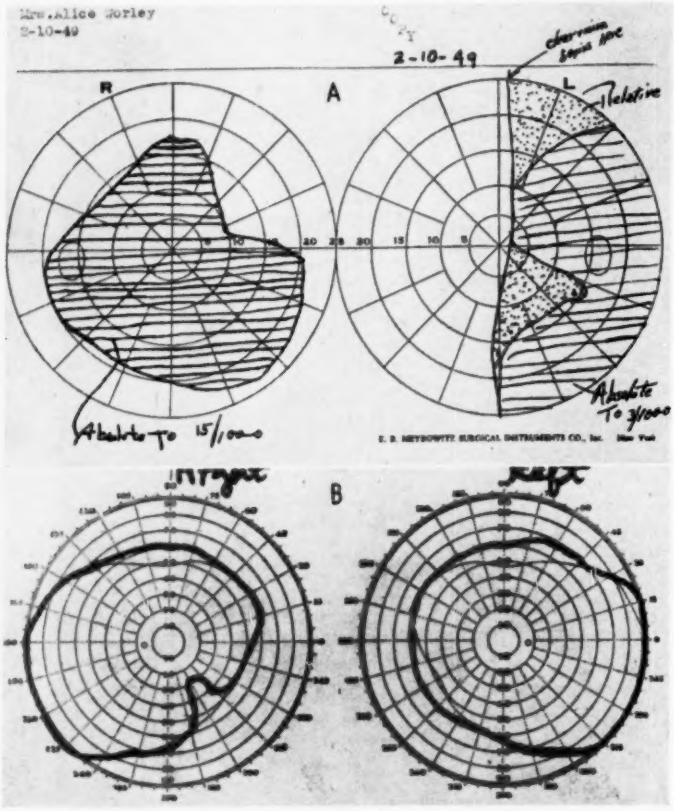

Fig. 24.—Visual fields of the patient A. W. before and after the operation.

A. W., a patient of Dr. Hamlin, a 28-year-old woman, complained of cessation of menstruation for five years and of rapidly failing vision for two months. At operation, in February, 1949, a cystic tumor was found and emptied and a drainage established. Before the operation one eye was practically blind, owing to a large central scotoma. The other eye had temporal hemianopsia. The eyes improved rapidly. Visual acuity and fields became and remain normal. The microscopic report was craniopharyngioma.

C. M., a 3½-year-old girl, was referred to Dr. Hamlin because of loss of vision. The sella was enlarged and located over a spongy sphenoid bone. The sphenoidal sinus was not developed. Two neurosurgeons hesitated to operate and suggested a transsphenoidal operation.

I operated, with the patient under ether anesthesia, and reached, by the way of submucous resection, a shallow sphenoidal cell, and kept on deepening the groove by chisel and curettes. These instruments did not prove to be the proper ones to achieve the goal. New instruments had to be chosen. It took four sessions, with two hours' anesthesia each time, before I reached the tumor by means of a specially adapted dental machine with special burrs. The progress was checked by roentgenograms.

During these operations and treatments, I enjoyed the assistance of Dr. Hamlin. Through a tunnel in the sphenoidal body the tumor was reached and exposed. The operative field measured 1.5 cm. in diameter and contained fluid, of which 5 cc. was saved. The vision of the child and her psychological adjustment improved remarkably. Three months after the operation the check-up examination revealed the broad tunnel in the sphenoidal body had closed. Although the vision was still improved, I opened the sphenoidal body again by burrs, reached the cyst, incised

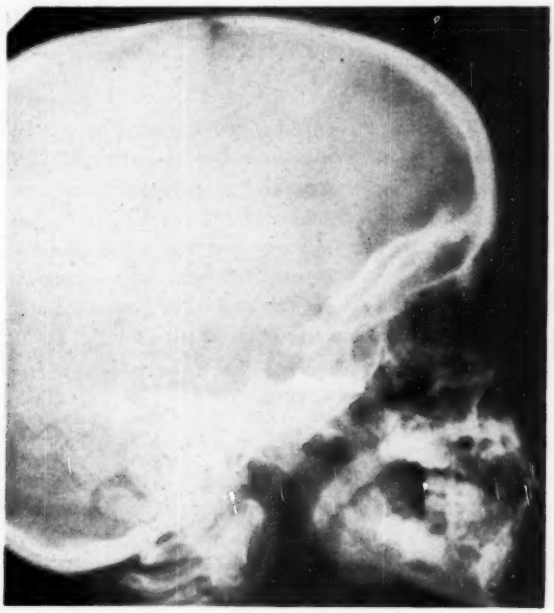

Fig. 25.—Roentgenogram showing tantalum tube introduced into the craniopharyngioma of a 3½-year-old girl, June, 1949, which is tolerated up to date.

it, and introduced a tantalum tube through the sphenoidal bone into the cyst. The child's condition was checked late in March, 1951. The tube was in correct position and was patent. The vision showed the improvement achieved after the first opening of the cyst. She attends kindergarten and competes nicely with other children, including her twin brother.

I selected from the 37 patients I have operated on in Boston those with conspicuous results, in order to show what can be achieved if the patient is operated on in due time, or at least not too late after the onset of symptoms. Whether or not the patient comes in due time to operation depends to some extent on the patient, but often on the ophthalmologist who may first see the patient.

The patients operated on in Boston in the last six years have been followed up for this short period only, and do not contribute conclusively to the evaluation

of lasting results. One must turn to the past to find out about the achievement of this method in regard to lasting results.

LASTING RESULTS

I have previously reported a follow-up study of patients treated by the described method in 1921.[73] I mentioned in this report that many patients could not be followed up because World War I had separated me from my patients and vice versa. This report included the period of trial 1910 to 1914, which accounts for the higher mortality in this report than in the subsequent reports of 1930[74] and 1932.[75]

In December, 1937, on the occasion of the International Ophthalmological Congress in Cairo,[76] I gave a review of the *277* patients operated on from 1919 to 1937, inclusive, a period of 19 years (Table).

The figure of *277* comprises patients from the age of 5 to *72* years, acromegalics with or without visual disturbance, 1 dwarf, 1 giant, 1 patient with Cushing's syndrome, and patients with the ophthalmic syndrome. The ophthalmic syndrome, i. e., visual disturbance, without conspicuous physical change, prevails.

Approximately 67% of these patients had solid chromophobe tumors; 19%, cystic degenerated chromophobe tumors; 8.5%, cystic craniopharyngiomas, and 8%, eosinophile tumors; one had glioma and one malignant adenoma (proved microscopically). Clinically, malignant adenomas were often observed. They manifested themselves by rapid and frequent recurrences. Patients with sellar decompression only and patients whose microscopic findings are not available are not included in this estimate.

COMPARISON OF RESULTS OF TREATMENT BY TRANSSPHENOIDAL AND CRANIAL METHODS

This review of *277* patients operated on during 1919 to 1937 is better evaluated if compared with the accomplishments of the cranial method. Henderson's review of Cushing's series reveals similar results with the transsphenoidal method. Of Cushing's 336 patients with pituitary tumor, *277* were operated on by the transsphenoidal, and 101 by the transfrontal, method. The mortality rate was in both series the same: about 5%. Cushing had used during 1913 to 1929 mostly the transsphenoidal method and during 1929 to 1932 the transfrontal method. The review was made in 1937. Therefore the length of time for observation of lasting improvement is different for the two methods. The longest-lasting improvements were cited for two patients operated on by the transsphenoidal method. This difference in the period of observation of the results for each method makes the

73. Hirsch, O.: Handbuch der speziellen Chirurgie des Ohres und der oberen Luftwege, Ed. 3, 1921, Vol. 3, p. 613.

74. Hirsch, O.: Über Hypophysentumoren und deren Behandlung, Klin. Monatsbl. Augenh. **85**:609, 1930.

75. Hirsch, O.: Die nasalen Operation der Hypophysentumoren, Klin. Monatsbl. Augenh. **89**:782, 1932.

76. Hirsch, O.: Diagnose und Therapie der Hypophysentumoren, in Transactions of the Fifteenth Concilium Ophthalmologicum Egypt **4**:119, 1937. Cairo, Vol. 12, p. 119, 1937.

Summary of Data on 277 Patients Operated on for Hypophyseal Tumors, 1910-1937

Patients operated on		277
Deaths after operation		13
Mortality rate		4.7%
Deaths after second operation		2
Mortality rate, including secondary operation		5.4%
Patients dying in 1921-1937		68
Patients dying 10-14 yr. after operation		8
After 14 and 10 yr. at ages 75 and 70	2	
After 10 yr., of carcinoma of breast and maxilla	2	
After 13 yr., of "grippe"	1	
After 11 yr., of sepsis following chiropody	1	
After 10 and 11 yr., of recurrent tumor	2	
Patients dying 6-7 yr. after operation		6
After 6½ yr., at the age of 75	1	
After 6½ yr., of "grippe"	1	
After 6½ yr., of "intracranial hemorrhage"	1	
After 6 yr., of diabetes plus retinitis	1	
After 6-7 yr., of recurrent tumor	2	
Patients dying 4-5½ yr. after operation		11
By suicide	1	
Of malignancy of the tumor	5	
Of unknown causes	5	
Patients dying 2-4 yr. after operation		13
Of malignancy of the tumor	13	
Patients dying within 2 yr. after operation		30
After a second operation by the endonasal method	2	
By a streetcar accident	1	
Of, "intracranial pressure"	1	
By malignant tumor	26	
Patients surviving operation		262
Patients followed up more than 10 to 19 yr. after operation		39
With lasting improvement	23	
With decreasing vision	6	
Without report for 2, 5, 6, 7 (2 patients), 9, 11 yr. after operation	7	
Without report	3	
Patients followed up more than 8 yr. after operation		26
With lasting improvement	17	
Achieving no improvement	2	
Able to work for 6 yr., then recurrence	1	
Without report for 4 yr	1	
Without report	5	
Patients followed up more than 5 yr. after operation		63
With lasting improvement	43	
With same condition as before operation	5	
Without improvement—decreasing vision	1	
With a second operation (craniotomy)	1	
With recurrences	7	
Without report	6	
Patients followed up to 4 yr. after operation		66
Patients with lasting improvement	41	
Patients with same condition as before operation	10	
Patient with improvement after a second transsphenoidal operation	1	
Patients with temporary improvement—then recurrence	4	
Patient with improvement but died of "apoplexy"	1	
Patients without improvement—decreasing vision	4	
Patients without reports	5	

SUMMARY OF REVIEW IN DECEMBER, 1937

Patients alive 4-19 yr. after operation		128
Patients with lasting improvement		83 (65%)
Patients with lasting improvement until end of life		12 (8.6%)
Total no. with improvement over 4-19 yr		95 (73.6%)
Death 10-14 yr. after operation	8	
Death 6-7 yr. after operation	3	
Death 5 yr. after operation	1	

(Causes of death were old age, accidents, and intercurrent disease)

In this analysis are not included six patients with inconclusive report 5 to 11 years after operation, but none since, seven patients alive 4 to 8 years with the same vision as before the operation, six patients alive more than 10 years, with decreasing vision, and one patient who reported that he was able to work for 6 years, then had decreasing vision. With any of these categories added, the percentage of success would rise (from 73.6 to 85).

comparison difficult. In addition, Cushing's work belongs to history. The current results of the cranial method must be dealt with. The mortality for the cranial method is 11.2% (Grant [51]). There are no better results published thus far (Horrax,[77] 13.3%; Olivecrona,[78] 11.7%). These figures do not include cases of craniopharyngioma, in which there is an operative mortality of 35%.

LATE RESULTS

I take for comparison Olivecrona's series [78] because they are in number and in length of observation analogous to mine.

Olivecrona's Transfrontal Operations	Hirsch's Transsphenoidal Operation
Of 291 patients 34 died after operations, i. e., 11.7%	Of 277 patients 15 died, i. e., 5.4%
After 2 to 18 years 130 patients were alive after operation	Of 128 patients who were alive 4 to 19 years after operation, 83 showed lasting improvement, i. e., 65%
Fully able to work were 94 patients, i. e., 54.5%	

In the experience of all neurosurgeons, as well as in my own, the period of two-year observation after operation is too short for judging late results. Most of the recurrences and deaths due to them happen during the first four years. I find in Table 3 of Olivecrona's series that the loss of patients within the subsequent two years is 5.3%. Deducting this percentage in order to place Olivecrona's and my review on the same time level, i. e., 4 to 18 and 4 to 19 years, respectively, I find that lasting results ("able to work") were achieved in 49.2% by the cranial method and in 65% by the transsphenoidal method.

In this 65% are not included 12 patients who enjoyed improvement of vision 5 to 14 years, until they died of intercurrent diseases. With these added, the percentage of lasting results would increase to 73.6 (of 140 patients, 95 had lasting results)—(see summary of review, p. 297). The term "lasting improvement" is based on the visual findings (acuity of vision and visual fields).

Lately, the pituitary tumors were separated into those with and those without cerebral extension, and the mortality rate was figured for each group separately. As a rule only pituitary tumors without visual disturbances, i. e., 50% of cases with acromegaly, have no cranial extension. All pituitary tumors with visual disturbances have such an extension. The size of such cranial extension, generally speaking, is in proportion to the visual disturbances. The more advanced these disturbances are, the larger is the extension. Patients with short optic nerves (about 6% of all patients) are exceptions to this rule. In such patients even a small tumor is able to produce severe visual disturbances. What should be called a cranial extension is left to arbitrary judgment. Consequently, the mortality rate figured on this basis could become arbitrary too. Since tumors with cranial extension are regularly found post mortem and are usually larger than anticipated before the operation, the impression has been established that the succumbing patients are those with the cranial extension, surviving patients those not having such an extension. In my review I have not made such differentiation, though a considerable number of patients operated on had what is now called intracranial extension.[20]

77. Horrax, G.: The Chromophobe Pituitary Adenomas: Surgical and Radiation Treatment, S. Clin. North America **31**:877, 1951.

78. Bakay, L.: Results of 300 Pituitary Adenoma Operations (Prof. Herbert Olivecrona's Series), J. Neurosurg. **7**:240, 1950.

LONG-LASTING IMPROVEMENT FROM 20 TO 30 YEARS

Since my last review of December, 1937, it has been impossible, due to World War II and postwar conditions, to contact the survivors. Therefore it is useless today to present any statistics on long-surviving patients. I tried to get in touch with my patients or with the physicians of the patients whom I had operated on before 1930, in order to find out about their condition. To my great surprise, I

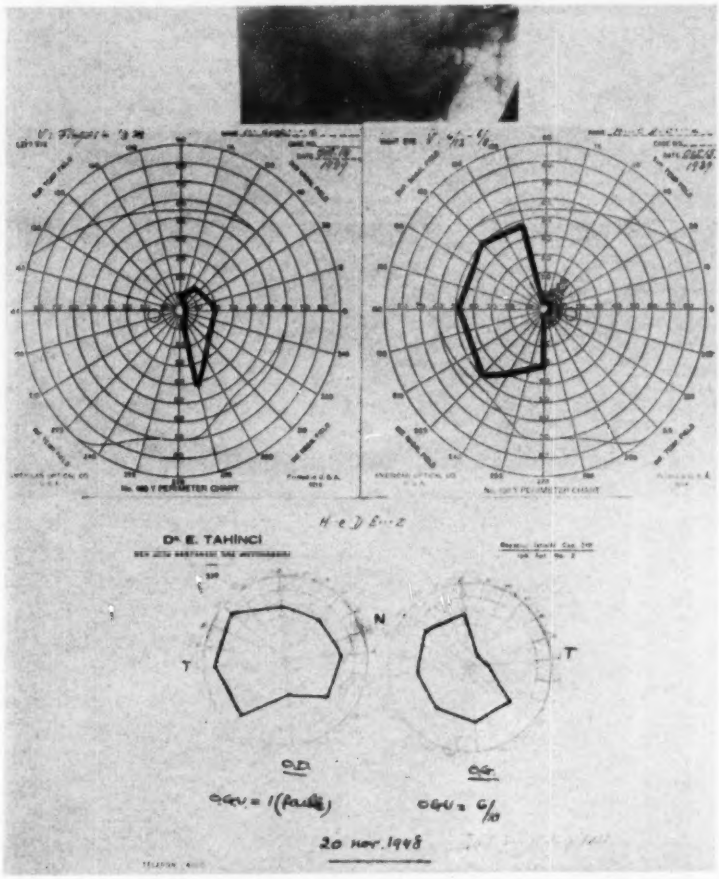

Fig. 26.—Roentgenogram and visual fields of one of the 19 patients with long-lasting results. This patient was operated on at the age of 33 for a cystic degenerated chromophobe tumor. The improvement due to operation plus radium treatment lasts up to date, 22 years after the operation.

have been able to obtain reports of 19 patients who have lived or were still living 19½ to 30 years after the operation. Of these 19 patients, 17 were operated on because of impaired vision, 2 of them for acromegaly; 15 maintained improvement of vision for 19½ to 30 years. I do not claim that this group is necessarily representative of all patients operated on before 1930; on the other hand, these are not merely my favorable patients, but represent all the survivors of at least 19½ years on whom follow-up reports were available.

A brief history of one case which seems to me of special interest follows: To my knowledge, the patient is the longest survivor of a pituitary-tumor operation

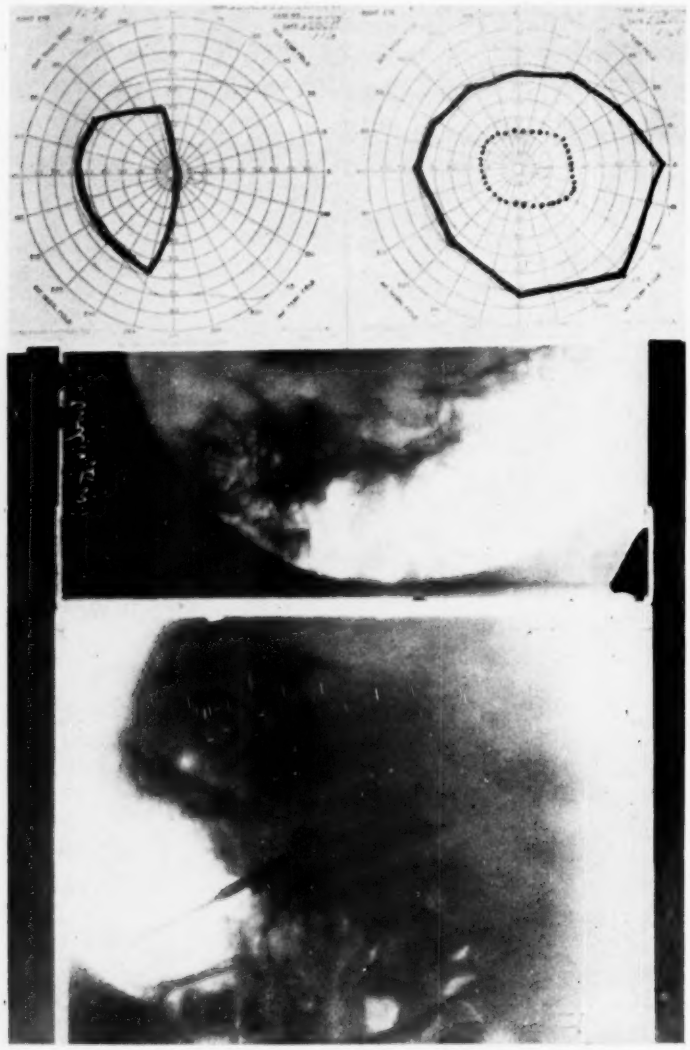

Fig. 27.—Fields of vision, and roentgenogram showing radium applicator in the patient A. Z. O., operated on and treated January, 1921. Improvement has lasted 31 years.

and had been previously operated on by the transfrontal route by Harvey Cushing. He is living and has been well for 30 years.

A. Z. O. (K.), a 27-year-old man, was operated on March 24, 1917, for a chromophobe tumor by the transfrontal way by Harvey Cushing. Cushing explained to me in a letter the choice of the method: "My reason for doing a transfrontal operation in this case, rather than a trans-

sphenoidal operation, which I prefer, was due to the fact that he has apparently a relatively small sella turcica, so that I felt that it would be somewhat difficult of access from below. You possibly may not find this could be the case."

Cushing's operation led to complete restoration of vision, even of the practically blind eye. After one year the vision started to fail again, and this time the left eye became blind. At the time the patient came to see me in Vienna the right eye had temporal hemianopsia and visual acuity of 5/6 and the disk was normal. The left eye was completely blind with atrophic excavation of the optic disk. The sella was moderately enlarged. I performed a sellar decompression, Jan. 26, 1921; i. e., I removed the floor of the sella but did not take any specimen for biopsy, because the nature of the tumor was known by previous operation. Radium was applied by the transsphenoidal method, first in an average dose. Since no results became apparent, the dose was doubled and tripled. At the end of the treatment visual acuity in the right eye was 5/5, and the field started to gain in the center. The left eye remained blind. During the following months after the radium treatment was ended, the patient regained a full field and normal acuity in the right eye; no other treatment was used. The patient married in 1929 and now has two healthy sons. In October, 1950, he was seen by the same ophthalmologist who had seen him in Vienna before the operation in 1921, and who is now in New York (A. Fuchs). The visual acuity in the right eye was 6/6; the field of vision for white and color were full; the left eye remained blind. This very good result lasts to the present time, 31 years after the transsphenoidal operation plus radium treatment. The patient is active in his very strenuous business.

The reason that I restricted myself to a sellar decompression, without removal of the tumor, was that after such an operation radium can be applied within a few days without risk of infecting the tumor. The radium application would have been delayed for at least six weeks if I had operated on the tumor. Besides, according to the information given me by Cushing, the tumor was a solid adenoma, which could not have been removed completely by any method. The tumor was very radium-resistant, and therefore the large dose had to be applied. The blind, left eye, showed a pale disk with atrophic excavation; that may be the reason that the left eye did not recover. Pallor of the disks—as I mentioned before—does not necessarily lead to a pessimistic prognosis, since a full restoration of vision is possible. Pallor of the disk is not identical with atrophy of the fibers (Walker and Cushing [38]). Before the first operation by Cushing, the disk of the left eye showed only slight pallor. At the time the patient came to see me, the left disk showed pallor plus atrophic excavation. Therefore the complete restoration after the first operation is in agreement with the above comment.

INDICATIONS FOR TREATMENT

One must decide practically between x-ray and operative treatment—and especially which of the two should be used first. With cystic tumors and craniopharyngiomas operation is the treatment of choice. Patients with acromegaly without visual disturbances react oftener favorably to medical and x-ray treatment, and only moderate enlargement of the sella presents a deterrent to transsphenoidal surgery. Cranial surgery too, could meet difficulties in this situation because the tumor lying beneath the diaphragm is not visible in the cranium.

In the case of solid tumors with progressive visual loss not yet progressed to bitemporal hemianopsia, x-ray treatment can be given preference, and operation can follow if this is without success. If the disturbance of vision has reached the stage of bitemporal hemianopsia, I should advocate that the operation be considered first, even if acuity of vision has not suffered greatly. On the other hand,

if acuity of vision has declined rapidly and extensively or if a central scotoma has developed, the same plan is valid in order to achieve a prompt decompression. In the indication for the operation one has to be directed not only by the charts which reflect the loss of visual fields, but also by a consideration of the mechanism which leads to the loss of vision. This mechanism is the compression of strangulation of the chiasm by the tumor and anterior communicating artery (Fig. 2). The latter cuts into the substance of the chiasma or of the optic nerves until nothing is left but the sheaths of connective tissue. In this advanced stage there is but little chance of recovery. Therefore, in the advanced stage of visual disturbance, decompression, which is achieved by operation in the quickest and surest way, is imperative.

To offer surgery when the patient is in imminent danger of going blind will jeopardize the chances of recovery.

One has to consider the individual circumstances of the patient—for instance, whether or not he is available at any time. Six weeks or longer must pass before any benefit from x-ray treatment can be demonstrated, which may be too long a time to wait for relief before deciding upon an eventual operation. Such an arrangement would meet with difficulties if the patient's home is very far away.

Important consideration must be given to the operative risk before deciding on whether to use x-ray treatment or surgery first. Radiation therapy involves practically no mortality, but does have some degree of hazard in threatening deleterious effects on normal tissues surrounding the target. The choice of the sequence of treatment depends on the estimation of risk and the probability of maximum benefit. The patient should be informed about the chances and the risk of the x-ray and operative treatment.

X-ray treatment results in practically no fatality, but offers a limited chance of improvement, as shown in the tabulation (page 282).

Cranial operation has a mortality of 11.2 to 13.3%, with chances of lasting improvement amounting to 49%.

Transsphenoidal operation has a mortality of 5%, with chances of lasting improvement amounting to 73.6%.

In conclusion, the question as to the primary use of operation or irradiation can be answered with respect to the transsphenoidal method thus: The operation should precede x-radiation or radium treatment. Such sequence of treatment would fulfill the logical plan of decompressing the optic pathways first and then preventing the regrowth of the tumor, if necessary, by irradiation.

EXPERIENCES OF RHINOLOGISTS AND SURGEONS WITH TRANS-SPHENOIDAL-SEPTAL METHOD

The endonasal method has been adopted by a number of rhinologists and surgeons in various countries. Most of them have used the septal method, as I have described, but some of the surgeons started the operation with a sublabial incision, as Loewe [79] suggested and Halstead [61] used for the first time.

Among the rhinologists who operated on pituitary tumors by the endonasal septum method were Loewe,[79] Spiess,[80] Holmgren,[81] Graham,[82] Hill,[83] Halle,[84] West,[85]

79. Loewe, L.: Zur Chirurgie der Nase, Berlin, Coblentz, 1905; Über die Freilegung der Sehnervenkreuzung und der Hypophysis, Ztschr. Augenh. **19**:456, 1908.

Sauer,[86] McBean,[87] Mayer,[88] Neumann,[89] Halsted,[90] Coffin,[91] Davis,[92] Barnhill,[93] Stein,[94] Sewall,[95] Lewis,[96] Knutson,[97] Killian,[98] Pifft,[99] Luescher,[100] Howarth,[101] Sargent,[102] Kutwirt,[103] Kutepoff,[104] Jentzer,[105] Kostič,[106] Sourdille,[107] Claus,[108]

80. Spiess, G.: Tumor der Hypophysengegend auf endonasalen Wege erfolgreich opereirt, München med. Wchnschr. **58**:2503, 1911; Heilung eines Falles von Meningitis nach endonasalen Operation eines Hypophysentumors durch Trypaflavininfusionen, Deutsche med. Wchnschr. **46**:207, 1920.

81. Holmgren, G.: Über einen Fall von Hypophysentumor erfolgreich nach Hirsch operiert, Ztschr. Ohrenh. **66**:39, 1912.

82. Graham, C. I.: Section of Laryngology (Exhibition of Cases and Specimens), Lancet **1**:242, 892, 1913.

83. Hill, W., in Transactions of the Seventeenth International Congress of Medicine, London, 1913, Sect. 15, Rhino-Laryngology, Part I, London, Oxford University Press, 1913, p. 189.

84. Halle, M., in Transactions of the Seventeenth International Congress of Medicine, London, 1913, Sect. 15, Rhino-Laryngology, Part I, London, Oxford University Press, 1913, p. 189.

85. West, J. M., in Transactions of the Seventeenth International Congress of Medicine, London, 1913, Sect. 15, Rhino-Laryngology, Part I, London, Oxford University Press, 1913, p. 189.

86. Sauer, W. E.: The Endo-Nasal Route of Attack in Hypophyseal Cases, Ann. Otol. **23**:971, 1913; **39**:66, 1930.

87. McBean, G.: Hypophyseal Tumor from the Rhinologist's Point of View, Internat. Zentralbl. Laryng. **29**:294, 1913.

88. Mayer, O.: Demonstration of a Case of Pituitary Tumor Operated by Hirsch's Method, Wien. klin. Wchnschr. **27**:914, 1914: Hypophysenoperation nach Hirsch vor einem Jahre-Radium Bestrahlung: dauernde Besserung Sehvermögens, Zentralbl. Hals-, Nasen- u. Ohrenh. **11**:48, 1927-1928; A Spontaneous Recovered Case of Liquor Fistula Following Hypophysis, Excerpta Med. **2**:532, 1948.

89. Neumann, H.: Zwei Fälle von operierten Hypophysentumoren, Wien. klin. Wchnschr. **27**:914, 1914.

90. Halsted, T. H.: Endo-Nasal Operation in Tumor of the Hypophysis: Report of a Case in a Female 9 Years of Age, Ann. Otol. Rhin. & Laryng. **23**:693, 1914; Report of Progress of a Case of Pituitary Tumor Reported in 1914, Pacific M. J. **59**:248, 1916.

91. Coffin, L. A., in discussion of Halsted.[90]

92. Davis, E. D.: Report of a Case of Sarcoma of the Pituitary Body by the Killian-Hirsch Operation, Proc. Roy. Soc. Med. (Laryng. Sect.) **2**:57, 1914-1915.

93. Barnhill, J. F., in discussion on Halsted, T. H.: Progress of a Case of Pituitary Tumor Reported 1914, Tr. Am. Laryng. A., 1915, p. 317.

94. Stein, O. J.: Report of a Case of Hypophyseal Growth Through the Nose and Sphenoid, Laryngoscope **25**:159, 1915.

95. Sewall, E. C.: Operation on the Hypophysis According to Hirsch's Method, J. A. M. A. **65**:681 (Aug. 21), 1915.

96. Lewis, F. O.: Pituitary Tumor, Operation by Trans-Sphenoidal Route, Pennsylvania M. J. **21**:282, 1917-1918.

97. Knutson, E.: Fälle von Hypophysentumoren, Internat. Zentralbl. Laryng. **32**:362, 1916.

98. Killian, G.: Hypophysentumor, Laryng. Gessellsch. zu Berlin (March 1) 1918; Internat. Zentralbl. Laryng. **35**:20, 1919.

99. Pifft, O.: Operation der Hypophysentumoren, München med. Wchnschr. **661**:605, 1919.

100. Luescher, F.: Personal communication to the author.

101. Howarth, W.: Sellar Decompression for Pituitary Tumors, Internat. Zentralbl. Laryng. **37**:7, 1921; J. Laryng. Rhin. & Otol. **36**:141, 1921.

102. Sargent, P.: Pituitary Tumors Treated by Decompression, Tr. Ophth. Soc. United Kingdom **41**:312, 1921.

103. Kutwirt, O.: Hypophysencyste nach Cushing operiert, Zentralbl. Hals-, Nasen- u. Ohrenh. **5**:431, 1924; **10**:645, 1927.

(Footnotes continued on next page)

von Eicken,[109] Meyer,[110] Sercer,[111] Lund,[112] Wein,[113] Bassin,[114] Demetriades,[115] Patterson and Phillips,[116] Kubo,[117] Segura,[119] Bourguet and Tscherniac,[120] and Rawson.[121]

Among the surgeons and neurosurgeons who used my method or modifications of it were Cushing,[65] Landon,[122] Leriche,[123] Locke,[124] Frazier,[125] and Cavina.[126]

Among the surgeons, Cushing [65] was the first who performed the attack on pituitary tumors by the septal route, using Loewe [79] and Halstead's [61] sublial incision. Cushing's series of 227 patients operated on by this method was, next to mine, the largest.

Cushing [65] expressed himself thus: "Doubtless in the majority of cases suitable for surgical treatment, the preferred avenue of approach will continue to be an

104. Kutepoff, I. P.: Hypophysisgeschwulst, Zentralbl. Hals-, Nasen- u. Ohrenh. **4**:107, 1923-1924.

105. Jentzer, M.: Tumeur de l'hypophyse à développement intrasellaire: hémianopsie bitemporale; acuité visuelle 1/10; acromégalie; opération par voie endoseptale, Bull. mem. Soc. nat. chir. **1**:958, 1924.

106. Kostić, D.: About 3 Cases of Pituitary Tumors Operated by Trans-Sphenoidal Method (Serbian), Zentralbl. Hals-, Nasen- u. Ohrenh. **9**:27, 1926-1927.

107. Sourdille, M.: Deux cas d'opération de Hirsch-Segura pour tumeurs de l'hypophyse, Zentralbl. Hals-, Nasen-, u. Ohrenh. **8**:771, 1925-1926.

108. Claus: Hypophysistumor, Zentralbl. Hals-, Nasen- und Ohrenh. **8**:772, 1925-1926.

109. von Eicken, in discussion of Claus.[108]

110. Meyer, M.: Über Geschwülste der Hypophysengegend (Hypophysentumoren, Hypophysengangtumor, Hirnbasis, Cholesteatom), Würzb. Abhandl. Gesamtgeb. Med. **22**:127-147, 1925.

111. Sercer, A.: Hypophysentumoren, Zentralbl. Hals-, Nasen- u. Ohrenh. **10**:809, 1927.

112. Lund, R.: Reflexions sur le traitement chirurgical des tumeurs de l'hypophyse, Acta chir. scandinav. **59**:491, 1926.

113. Wein, Z.: Operierter Fall von Hypophysengeschwulst (perseptal nach Hirsch), Zentralbl. Hals-, Nasen- u. Ohrenh. **20**:636, 1933.

114. Bassin, R.: Ein Fall von operiertem Hypophysentumor (trans-septal), Zentralbl. Hals-, Nasen- u. Ohrenh. **25**:201, 1935.

115. Demetriades, T.: Pituitary Operation According to Hirsch, Zentralbl. Hals-, Nasen- u. Ohrenh. **28**:4, 1937.

116. Paterson, R.: Trans-Sphenoidal Decompression for Pituitary Adenoma by Gilbert Phillips (Sidney, Australia), Brit. J. Surg. **26**:242, 1938-1939.

117. Kubo, M.: Intracranial Pituitary Tumor Operated by Hirsch's Method, Zentralbl. Hals-, Nasen- u. Ohrenh. **32**:387, 1939.

118. Footnote deleted.

119. Segura, E. V.: Contribution à la chirurgie de l'hypophyse, Rev. oto-neuro-ocul. **1**:657, 1923.

120. Bourguet, J., and Tscherniac: Tumeurs de l'hypophyse, Bull. et mém. Soc. franç. opht. **38**:756, 1924; Zentralbl. Hals-, Nasen- u. Ohrenh. **9**:258, 1926-1927; **12**:591, 1928; **18**:34, 1932.

121. Rawson, R. D.: Tumor quistico de la hypofisis: Operacion de Hirsch-Segura, Rev. oto-neuro-oftal. **7**:8, 1932.

122. Landon, L. H.: The Role of the Rhinologist in Hypophyseal Surgery, Pennsylvania M. J. **19**:675, 1915.

123. Leriche, M., and Perrin: Acromégalie avec perte progressive de la vision: Trépanation decompressive de la selle turcique per voie endonasale médiane sous-muqueuse, Soc. méd. hôp. Lyon, Dec., 1920.

anterior transsphenoidal one." Cushing used this method from 1912 until 1929 regularly. In the following three years, until his retirement, in 1932, this method figured less often, according to Henderson's review.[127]

Another leader in American neurosurgery, Charles Frazier, of Philadelphia, also favored the trans-sphenoidal-septum method. In the discussion on Landon's paper,[122] Frazier expressed himself as follows, in 1915:

> At first I advocated the frontal method, but for the present I have abandoned this for the transsphenoidal method, because, with the latter, in the removal of the floor of the sella turcica we have the only permanent means of directly relieving pressure upon the optic tracts.

Nine years later, in 1924, Frazier [128] wrote: "Since Hirsch first proposed the transsphenoidal operation in 1910 there has been a continuous reduction in mortality, so that today the operation is devoid of serious risk." In 1926 Frazier [128] reported 53 cases of the transsphenoidal operation, with a mortality rate of 4.5%.

> The pituitary adenoma is a slow growing affair, and the expectation of life may cover many years. Hence the operation with least hazard must be given the preference. . . . In previous communications we have discussed the reasons which, for the time being, have determined our preference for the transsphenoidal approach. Suffice it to say here that recognizing its limitation and recognizing the element of risk is so much less by the transsphenoidal approach there should be no doubt to the mind of the operator alive to his responsibilities which method to select.

Dr. Norman M. Dott,[128a] neurosurgeon at the Royal Infirmary, Edinburgh, informed me that he had performed 60 trans-sphenoidal-septal operations, with two postoperative deaths.

On the other hand, lately, a neurosurgeon, known for his experience and masterly skill, James L. Poppen,[129] expressed his concern in regard to the results of the cranial operation on pituitary tumors: "One can by no means be smug about the results of treatment of pituitary tumors. The outlook is not so favorable as had been assumed before careful evaluation of the results."

In spite of the favorable experiences of the neurosurgeons with the transsphenoidal method and the quoted concern about the results by the cranial method, the transsphenoidal method has lately been neglected. An explanation comes from

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124. Locke, C. E., Jr.: The Trans-Sphenoidal Versus the Osteo-Plastic Cranial Approach for Pituitary Adenomas, Arch. Neurol. & Psychiat. **5**:749 (June) 1921.

125. Frazier, C. H.: The Surgical Management of Pituitary Lesions, Atlantic M. J. **29**:435, 1926.

126. Cavina, C.: Resultati immediati e lontani di 14 ipofisectomie per affezioni endosallari, Atti Soc. Ital. chir., 37th Congress, Rome, Oct., 1930; Zentralbl. Hals-, Nasen- u. Ohrenh. **18**:34, 1931.

127. Henderson, W. R.: Pituitary Adenomata: Follow-Up Study of Surgical Results in 338 Cases (Dr. Harvey Cushing's Series), Brit. J. Surg. **26**:811, 1939. In 1930 Cushing [19] reported a series of suprasellar tumors: meningiomas, craniopharyngiomas, and adenomas with visual disturbances but with normal or moderately enlarged sella. These suprasellar tumors (discussed above) were brain tumors and did not belong in the realm of the transsphenoidal operation; the patients were therefore operated on by the cranial method. The interpretation that Cushing deserted the transsphenoidal method cannot be upheld by means of any of Cushing's publications. It is apparently owing to the difference in material that the transsphenoidal method figured much less in the last years of Cushing's activity.

128. Frazier, C. H.: The Surgery of the Pituitary Body, Arch. Surg. **8**:39 (Jan.) 1924.

128a. Personal communication to the author.

129. Poppen, J. L., in discussion of Grant, F. C.: Surgical Experience with Tumors of the Pituitary Gland, J. A. M. A. **136**:671 (March 6) 1948.

one of the pioneers in American neurosurgery, E. Sachs,[130] who wrote that the neurosurgeons "are more at home in the cranial cavity."

The transsphenoidal method should be given equal consideration with the cranial operation and irradiation therapy in the treatment of pituitary tumors.

SUMMARY

Pituitary tumors are classified from the surgical point of view.

Basic symptoms for the diagnosis of pituitary tumors are enumerated.

The achievements and limitations of x-ray and operative treatments are briefly discussed.

The trans-sphenoidal-septum method used by me and the treatment of solid and cystic tumors by this method are described.

The value of additional radium treatment to prevent regrowth and to destroy and shrink the remnants of the tumor not radically removed is emphasized.

The operative mortality in the trans-sphenoidal-septum method is now 5% (including craniopharyngiomas) and is decreasing with the help of antibiotics.

Complications and their cure are discussed.

Immediate results in single cases of each group of intrasellar, craniosellar, and cystic tumors are presented.

A review of 277 cases followed up for 4 to 19 years is given.

Nineteen cases are mentioned in which the improvement has been maintained for 19½ to 30 years.

In conclusion, the transsphenoidal method has to be considered with regard to the low risk and long-lasting cures in cases of pituitary tumors with enlarged sella whenever operative treatment is indicated.

186 Commonwealth Ave.

130. Sachs, E.: Diagnosis and Treatment of Brain Tumors and Care of the Neuro-Surgical Patients, Ed. 2, St. Louis, C. V. Mosby Company, 1949, p. 292.

HERPES ZOSTER OF THE CEPHALIC EXTREMITY

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HERPES zoster, or zona, was well known to the ancients. It was not until the nineteenth century that herpes zoster was recognized as the cutaneous phase of a disease involving the sensory portion of the spinal nerves. Von Baerensprung in 1861 first demonstrated at autopsy inflammatory lesions in the posterior root ganglia. In 1900 the work of Head and Campbell [1] led to our current hypothesis of herpes zoster. The disease is now generally regarded as an acute specific infection caused by a neurotropic virus with a predilection for the posterior spinal ganglia or the extramedullary ganglia of the cranial nerves, conferring an immunity and running a definite clinical course accompanied with an erythematous vesicular eruption in the cutaneous or mucosal distribution of the sensory nerves.

Although many observers in the past century reported cases of herpes zoster of a cranial nerve, chiefly of Gasserian origin, but including herpes oticus and Gasserian herpes zoster associated with paralysis of the facial nerve, it remained for Hunt,[2] between 1906 and 1910, to present in a brilliant series of papers his concept of a posterior poliomyelitis of the geniculate, cochlear, vestibular, glossopharyngeal, vagal, and upper cervical ganglia. His main contribution is generally considered to be the much debated hypothesis of herpetic inflammation of the geniculate ganglion of the seventh cranial nerve, which syndrome bears his name. He also defined the clinical picture of herpes zoster of the cephalic extremity as we know it today: herpes facialis (Gasserian herpes); herpes oticus (geniculate herpes); herpes pharyngis (herpes zoster of the glassopharyngeal ganglia); herpes laryngis (herpes zoster of the nodosal ganglia); herpes occipitocollaris (herpes zoster of the upper cervical root ganglia).

It is stated in the literature that these conditions are not infrequently seen but are often not diagnosed. In view of an apparent increase in the incidence of virus diseases in general, and because several of the newer antibiotics offer some hope of specific therapeusis, a review of herpes zoster of the cephalic extremity from an anatomicoclinical point of view is thought to be timely.

Read before the Section on Laryngology, Otology, and Rhinology at the One Hundredth Annual Session of the American Medical Association, Atlantic City, June 13, 1951.

1. Head, H., and Campbell, A. W.: Brain **23**:353-523, 1900.

2. Hunt, J. R.: J. Nerv. & Ment. Dis. **34**:76-96, 1907; Symptom-Complex of Acute Posterior Poliomyelitis of Geniculate, Auditory, Glossopharyngeal and Pneumogastric Ganglia, Arch. Int. Med. **5**:631-675 (June) 1910.

HERPES ZOSTER

In general, an attack of herpes zoster is initiated by a prodromal period of three or four days. The patient feels ill; there is some elevation in temperature, and pain and hyperesthesia of the skin in an area innervated by the affected nerve are experienced. The appearance of the characteristic erythematous and vesicular eruption in the anatomical distribution of the peripheral sensory nerve is the diagnostic feature. Regional lymphadenopathy may be present. The process may last three to six weeks and may be followed, especially in elderly and in debilitated patients, by a painful postherpetic neuritis of several months' and even years' duration. There is often a moderate increase in cells, chiefly lymphocytes, in the cerebrospinal fluid. The pain of herpes zoster may be absent or may be severe, usually diminishing with the subsidence of the eruption, but leaving an annoying hyperesthesia in the affected area. Bilateral herpes zoster is a rare manifestation of the disease.

It is considered at present that the basic lesion of herpes zoster is probably an inflammatory disease located in the dorsal root or cranial sensory nerve ganglia. These specific pathological changes are now regarded as diffuse rather than localized, extending to involve not only the ganglion but also the posterior and anterior horn cells and the meninges. The cutaneous or mucosal vesicle is thought to be a result of the peripheral migration of the virus from the ganglionic focus to a location in the cutaneous and/or mucosal epithelial cells; a virus has been shown to be present in the vesicle. The presence of acidophilic intranuclear inclusion bodies in the cells about the vesicle indicates the activity of the specific virus.

Lever [3] summed up the histopathological features of herpes zoster as follows:

> The virus of herpes zoster is a neurotropic as well as an epidermotropic virus. In the neural segment corresponding to the cutaneous eruption, one finds: (1) severe inflammation of the ganglion, (2) peripheral mononeuritis, not only distal to the ganglion but also in the anterior and posterior nerve roots, (3) a unilateral, segmental poliomyelitis and (4) localized leptomeningitis.[4]

HERPES FACIALIS

By far the commonest of the sensory cranial nerves affected by the herpetic process is the trigeminal nerve, particularly the ophthalmic division (Fig. 1). Herpes ophthalmicus, in addition to its characteristic painful manifestations and diagnostic cutaneous eruption, is complicated in more than 50% of cases by serious ocular involvement in which every global structure may be affected. The seat of the infection lies in the Gasserian ganglion; the most commonly involved portion is the cells designated to the frontal nerve. As has long been pointed out, if the nasociliary nerve is implicated, as evidenced by lesions on the forepart and tip of the nose, ocular extension is more probable. Palsies of the extraocular muscles are somewhat uncommon. The other branches of the trigeminal nerve are involved less frequently. Figures 2 and 3 illustrate two cases of Gasserian herpes limited to the mandibular division. Cases have been reported in which the eruption was limited to the distribution of the first and third divisions of the fifth cranial nerve, as well as to that of the first and second divisions. The Gasserian zoster area is

3. Lever, W. F.: Histopathology of the Skin, Philadelphia, J. E. Lippincott Company, 1949.

4. Denny-Brown, D.; Adams, R. D., and Fitzgerald, P. J.: Pathologic Features of Herpes Zoster: Note on "Geniculate Herpes," Arch. Neurol. & Psychiat. **51**:216-231 (March) 1944.

Fig. 1.—Herpetic involvement of ophthalmic division of trigeminal nerve.

Fig. 2.—Gasserian herpes limited to mandibular division.

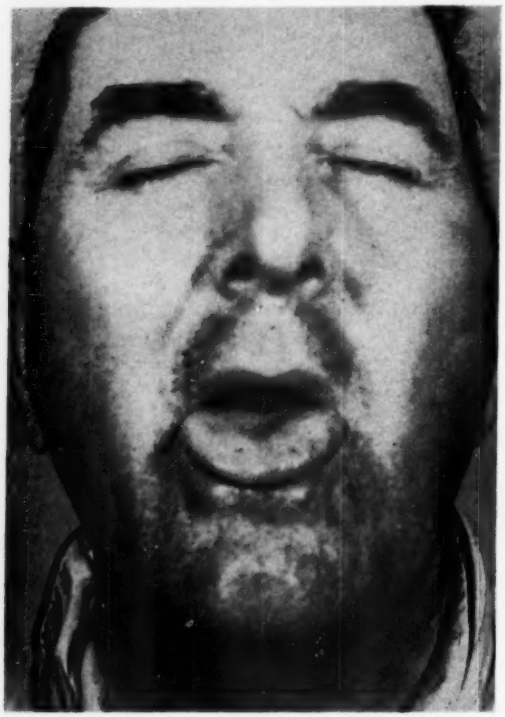

Fig. 3.—Gasserian herpes limited to mandibular division.

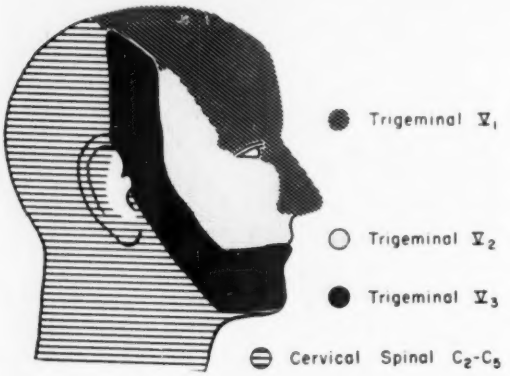

Fig. 4.—Sensory nerve distribution to head and neck in Gasserian zoster area.

illustrated in Figure 4. Occasionally all three branches of the trigeminus are simultaneously affected, and rarely bilateral involvement has been described. Gasserian herpes zoster has been reported to be associated with simultaneous involvement of other cranial nerves in the following manner.

1. Gasserian herpes zoster with paralysis of the facial nerve
2. Herpes ophthalmicus and herpes oticus with or without facial paralysis and herpes pharyngis
3. Gasserian herpes zoster and geniculate herpes zoster with facial and oculomotor paralysis
4. Gasserian herpes zoster with facial paralysis and with cochlear and vestibular symptoms
5. Herpes zoster involving the 5th, 7th, 9th, and 10th nerves with paralysis of the intrinsic laryngeal and facial muscles

As can be seen, many and variable combinations of herpes zoster are possible.

HERPES OTICUS

The most controversial of Hunt's views revolves about his ideas concerning herpes oticus. Hunt contended that the syndrome of otalgia plus a herpetic eruption appearing on a certain area of the external ear was due to a posterior poliomyelitis of the geniculate ganglion of the facial nerve. Depending upon the severity and the extent of the disease, the process may involve the seventh and eighth nerves in a varying degree, as proposed by Hunt in the following classification:

1. Herpes oticus (without neurological signs)
2. Herpes oticus with facial paralysis
3. Herpes oticus with facial paralysis and auditory symptoms
4. Herpes oticus with paralysis of the facial nerve and Ménière's symptom complex

Hunt stated that each of these subdivisions had been separately described by previous observers, but their common pathological features had not been heretofore recognized. The clinical entity, herpes oticus, therefore, involves the sensory portion of the seventh cranial nerve, the sensory cells of which arise in the geniculate ganglion and extend centrally via the nervus intermedius. The disease is characterized by a history of pain in the affected ear, preceded by the prodromal symptoms of malaise and fever and followed by a vesicular eruption in the geniculate zoster zone of the auricle (Fig. 5). When the ganglion alone is involved, herpes oticus results. In isolated herpes oticus there may be only a few vesicles in the concha or in the external auditory canal, or the skin of the auricle may be erythematous and swollen, resembling acute external otitis. Hunt thought that paralysis of the facial nerve resulted from pressure of the inflamed ganglion or from direct extension of the herpetic inflammation to the nerve trunk (Figs. 6 and 7). Simultaneous involvement of the auditory and vestibular ganglia, either as the original site of infection or from geniculate extension, produces the auditory symptoms of tinnitus and deafness or the auditory and vestibular signs of Ménière's symptom complex. Figure 8*A* illustrates schematically the communicating branches between the various components of the seventh and eighth nerves.

Despite valid objections to some of Hunt's theories, and despite the lack of histopathological proof or disproof of herpetic inflammation confined to the geniculate ganglion, the syndrome of herpes oticus has been generally accepted. From a clinical point of view it is frequently possible, from the herpetic localization and symptomatology and with the use of the topognostic analysis of Tschiassny,[5] to evaluate the anatomical extent of the inflammatory process.

The geniculate zoster area was described by Hunt as an irregular cone-shaped zone, including the whole or parts of the tympanic membrane, external auditory canal, concha, anthelix, fossa of the anthelix, tragus, antitragus, and upper part of the lobule. As illustrated in Figure 8*B*, this geniculate zoster zone on the external

Fig. 5.—Vesicular eruption of herpes oticus in geniculate zoster zone of auricle.

ear roughly covers, with some overlapping, the middle third of the ear, bounded anteriorly by the zoster zone of the third division of the fifth nerve and posteriorly by an area supplied by the vagus and upper cervical nerves. It can be seen, therefore, that the skin of the external ear is supplied by the 5th, 7th, 9th, and 10th nerves and the 2d and 3d cervical nerves. In this connection the 9th cranial nerve supplies anastomotic branches to the 10th nerve and to the facial nerve. These branches of communication of the 9th nerve arise from the petrous ganglion and extend to the auricular branch of the vagus (Arnold's nerve) and to the jugular ganglion of the vagus nerve; the glossopharyngeal facial connection is both by way of a branch which arises from the trunk of the nerve below the petrous ganglion

5. Tschiassny, K.: Ann. Otol. Rhin. & Laryng. **55**:152-174 (March) 1946.

and joins the digastric branch of the facial nerve and by indirect connections via the tympanic plexus. Thus, the presence of auricular herpes necessitates a careful consideration of the zoster zone involved for differentiation between herpes oticus and vagoglossopharyngeal, Gasserian (mandibular), or cervical herpes.

The intraoral manifestations of herpes oticus are based on the palatal branches of the facial nerve via the greater superficial petrosal nerve and/or the chorda tympani nerve. The facial nerve supplies the main part of the soft palate with parasympathetic fibers, while the margins of the soft palate and uvula receive their sensory innervation from the glossopharyngeal nerve. It is thought that the chorda tympani nerve, or other filaments of the facial nerve, conveys some fibers of general

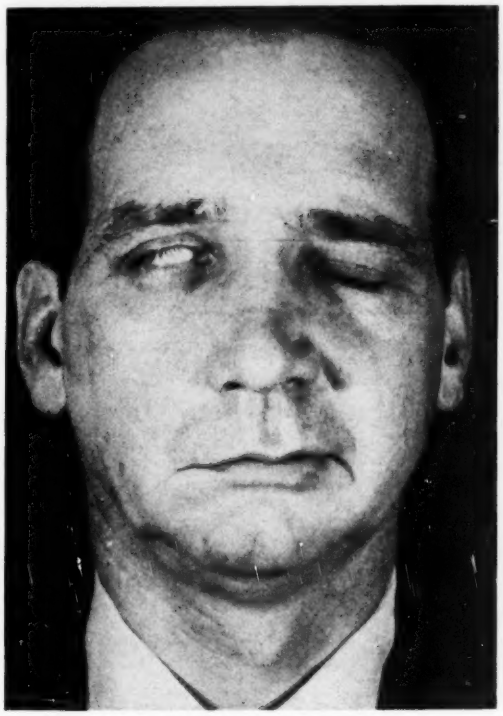

Fig. 6.—Facial paralysis in patient with herpes oticus.

sensation to the tongue and palate, as well as the special sensation of taste to the anterior two-thirds of the tongue. This pathway is used to explain the unilateral herpetic eruptions on the tongue in the distribution of the chorda tympani nerve in cases of geniculate herpes. In Figure 9 is shown a herpetic eruption on the tongue in a patient with paralysis of the facial nerve. In addition, intraoral lesions may arise from simultaneous herpetic infection of the 5th, 7th, 9th, and 10th nerves, the zoster zones of which are illustrated in Figure 10. The anterior two-thirds of the tongue is, therefore, a zoster area of the 5th and 7th nerves, and the base of the tongue is a zoster area of the 9th and 10th nerves.[5] It has also been suggested that the lateral margins of the anterior portion of the tongue are innervated by the glossopharyngeal nerve.

Fig. 7.—Herpes oticus: Herpetic vesicles in geniculate zoster zone of patient shown in Figure 6.

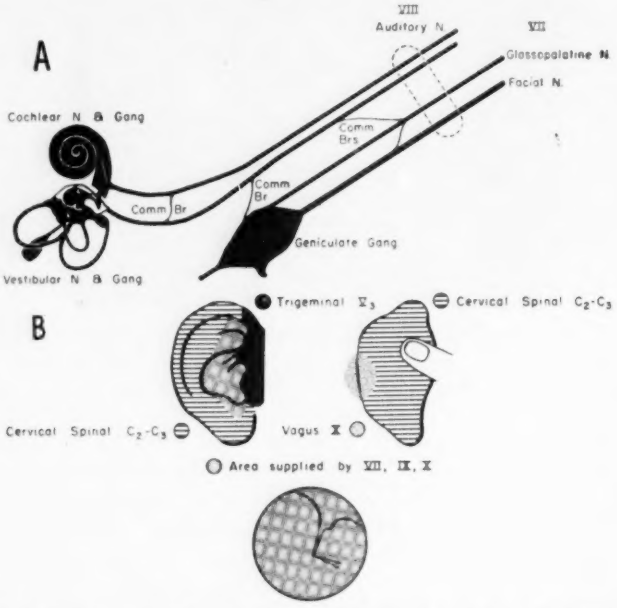

Fig. 8.—*A*, schematic diagram showing probable communications between facial and auditory nerves. (Modified from Cunningham's Textbook of Anatomy, Ed. 95, edited by J. C. Brash, London, Oxford University Press, 1951.) *B*, sensory nerve distribution to auricle, canal, and tympanic membrane.

By using the evidence produced by involvement of various branches of the seventh nerve (Fig. 11), Tschiassny [5] has given a clinical means of establishing the localization of the facial nerve lesion. The classification he proposed embodies an analysis of the sense of taste, the activity of the lacrimal gland, and the stapedius reflex. In the presence of a peripheral facial palsy the preservation of the sense of taste on the anterior two-thirds of the tongue on the same side and hyperlacrimation indicate a lesion peripheral to the branching of the chorda tympani nerve. In a lesion above this branch, the sense of taste is lost but hyperlacrimation is preserved. A transgeniculate lesion is evidenced by a loss of taste and absence of tears. In a lesion of the facial nerve central to the ganglion, the lacrimation

Fig. 9.—Herpetic eruption of tongue in a patient with paralysis of facial nerve.

function is lost but the taste function is unimpaired. The reason for this is the pathway of the taste fibers, which are said to extend from the geniculate ganglion by way of the greater and lesser superficial petrosal nerves to the tympanic plexus and are carried to the brain stem via the glossopharyngeal nerve. There are much confusion and difference of opinion concerning the exact pathway of taste fibers centrally from the anterior two-thirds of the tongue. The stapedius reflex is absent when the site of the lesion is above the branching of the stapedius nerve and is present when the site of the lesion is below this point. The absence of this reflex is detected by the symptom of auditory hypersensitivity. For this phenomenon frequently referred to as "hyperacusis," the more appropriate term "phonophobia" is suggested by Tschiassny.[5]

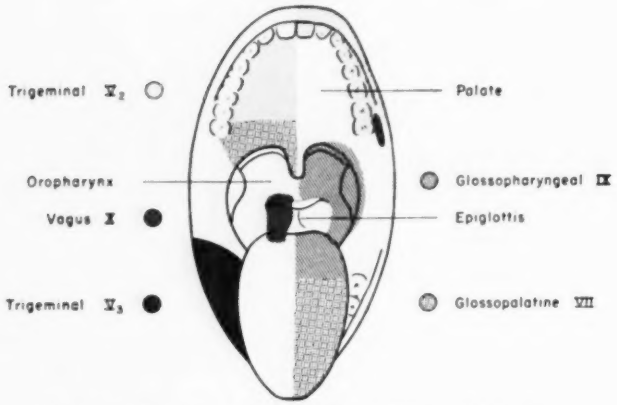

Fig. 10.—Sensory nerve distribution to intraoral structures.

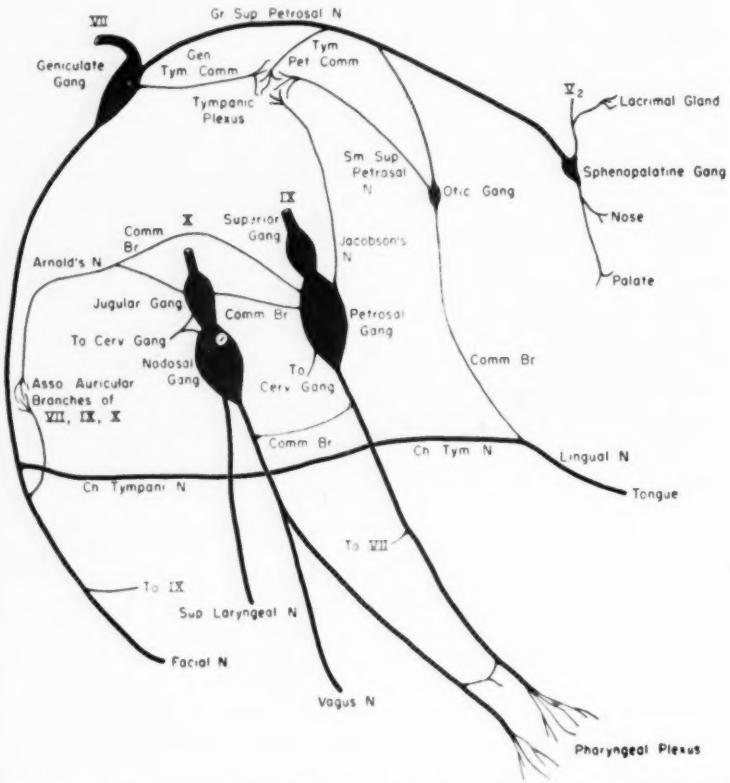

Fig. 11.—Schematic diagram showing sensory interrelationship between facial, glossopharyngeal, and vagus nerves.

It can be seen from Figure 11 that with this intricate overlapping network it is often impossible in some of the multiple manifestations of herpes zoster to designate one ganglion as the original herpetic focus, and this gives rise to the dispute whether the geniculate ganglion or some other ganglion is the fundamental site of infection in herpes oticus. It was postulated by Hunt that the lesion of herpes zoster predominantly centers in one ganglion; other ganglia above or below in the cranial-cervical ganglionic chain may show inflammatory changes, but of less degree. The zoster eruption may be seen in two separate and distinct zoster zones.

This hypothesis explains in part the cases reported in which herpes oticus is combined with simultaneous infection in the 8th, 9th, and 10th nerve ganglia.[6] Less well interpreted are the cases of Gasserian and cervical herpes zoster with facial paralysis and with cochlear and vestibular disturbances.

As additional clinical and pathological studies are presented, it can be anticipated that a clearer understanding of the term "Hunt's syndrome" will be reached. As indicated by Tschiassny,[5] the contribution of Johnson and Zonderman[7] is one in point.

Primary herpetic infection of the auditory and vestibular ganglia is rarely diagnosed. When acute cochlear and vestibular symptoms present themselves in association with herpes oticus or cervical herpes, the nature of the infection is readily recognized. If the deafness and vertigo are the initial symptoms, it is presumed that the eighth nerve ganglia are primarily involved. These ganglia are occasionally the sole site of the herpetic process, producing a mild to severe degree of tinnitus, deafness, or Ménière-like response. Herpes zoster of the eighth nerve should be included in the differential diagnosis of acute vestibular or cochlear episodes, especially if cerebrospinal fluid lymphocytosis is present.

HERPES PHARYNGIS

Herpes pharyngis, or herpes zoster of the glossopharyngeal nerve, long recognized by laryngologists, is perhaps uncommon in itself or frequently, as in herpes zoster of the vagus nerve, is not diagnosed. Hunt felt that the ganglia of the 9th and 10th nerves, being derived from the same embryologic structure as the posterior spinal, the Gasserian, the geniculate, and the auditory vestibular ganglia, could also be involved in the specific ganglionic inflammation of herpes zoster.

The glossopharyngeal ganglia have sensory representation on the auricle and the mucous membrane of the posterior margin of the soft palate, uvula, anterior and posterior pillars, posterior lateral surface of the tongue, tonsils, and adjacent pharyngeal region. The diagnosis is suggested by the pharyngeal pain and the strictly unilateral vesicular eruption and anesthesia in the glossopharyngeal zoster zone.

Reports in the literature of cases of herpes zoster of the glossopharyngeal nerve are not common.[8] More frequently glossopharyngeal herpes is diagnosed in conjunction with herpes zoster of the vagus nerve and in combination with geniculate herpes zoster and facial paralysis. The pharyngeal plexus is made up of the pharyn-

6. Rabbiner, M.: M. Rec. **148**:440-443 (Dec. 21) 1938.

7. Johnson, L. F., and Zonderman, B.: Herpes Zoster Oticus ("Ramsey-Hunt Syndrome"): Report of Case, Arch. Otolaryng. **48**:1-8 (July) 1948.

8. Wright, F. J.: Lancet **2**:946-947 (Dec. 27) 1947.

geal branches of the glossopharyngeal, vagus, and sympathetic nerves and provides the anatomical connection between these nerves. There are, as well, connections at the ganglionic level. When the vagus is involved, nausea and vomiting are often conspicuous symptoms.

HERPES LARYNGIS

The anatomical ramifications of the 10th cranial nerve provide the interesting features of the clinical entity, herpes laryngis, or herpes zoster of the vagus nerve. The sensory fibers arise from cells in the jugular and nodosal ganglia. There are connecting links between the vagus, the glossopharyngeal, the spinal accessory, the facial, the hypoglossal, and the upper two cervical nerves. The auricular branch of the vagus (Arnold's nerve) is given off in the jugular fossa and is soon joined by a filament from the petrous ganglion of the glossopharyngeal nerve. The auricular nerve leaves the skull via the facial canal, where it lies in contact with the facial nerve as far as the stylomastoid foramen. It supplies the zoster zone of the posterior portion of the auricular and adjacent mastoid area, the posterior segment

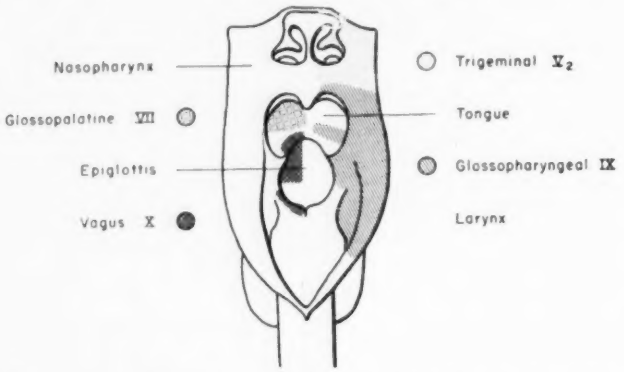

Fig. 12.—Sensory nerve distribution to nasopharyngeal, oropharyngeal, and hypopharyngeal structures (posterior view).

of the external auditory canal, and the posterior inferior surface of the tympanic membrane. The pharyngeal and superior laryngeal branches provide the zoster zone (Fig. 12) in the pharynx and larynx, an area covering the arytenoid eminences, the aryepiglottic folds, the pyriform sinus, the epiglottis, a wedge-shaped portion of the tongue in front of the epiglottis, and a portion of the soft palate. The pharyngeal branches of the vagus join the glossopharyngeal and sympathetic nerves and are the motor component of the pharyngeal plexus. The recurrent laryngeal branch is the main motor nerve of the larynx.

Clinically, herpes laryngis is characterized by unilateral hypopharyngeal and auricular pain with dysphagia. The vesicular eruption is limited to the vagal zoster area. Nausea and vomiting may be conspicuous features; bradycardia and hiccough may occur.

Owing to the intercommunicating network of cranial nerves and depending upon the degree and extent of the herpetic infection, the herpes laryngis may present itself in a varied pattern. As reported in the literature, the superior laryngeal

branch of the vagus may be involved in isolation [9] or with concomitant glossopharyngeal involvement.[10] One of us (G. S. F.) recently saw a case of laryngeal herpes with motor paralysis of the vocal cord on the same side. McKenzie [11] reported a similar case in 1915.

In the case reported by Negus and Crabtree [12] the herpes affected the geniculate ganglion of the seventh nerve, the petrosal ganglion of the ninth, and the nodosal ganglion of the vagus. The facial nerve involvement consisted of aural herpes, right facial paralysis, absence of taste on the right side (chorda tympani branch), palatal herpes, and absence of lacrimation on the right side (great superficial petrosal branch via the sphenopalatine ganglion). According to Tschiassny's classification, the lesion would be transgeniculate. Implication of the glossopharyngeal nerve was indicated by anesthesia of half of the pharynx and edema of the faucial pillars and part of the palate. The involvement of the vagus nerve was especially interesting, as evidenced by the auricular eruption (Arnold's nerve), paralysis of the pharyngeal constrictors on the right side (pharyngeal branch), herpetic vesicles on the epiglottis and the aryepiglottic fold (superior laryngeal branch), and paralysis of the right vocal cord (recurrent laryngeal branch). These authors also noted an immobility of half of the palate which was due to paralysis of the levator veli palatini muscle (pharyngeal plexus formed by the union of the 9th and 10th nerves).

In the four cases of herpes zoster reported by Engström and Wohlfart,[13] three were interpreted as of vagoglossopharyngeal involvement with auricular herpes. The fourth case was described as of the Hunt syndrome (herpes oticus, facial palsy, and acoustic lesion). The eighth nerve was implicated in all four cases; motor paralysis of the pharyngeal and laryngeal muscles was present in three patients and paralysis of the facial nerve in two. These cases are examples of the simultaneous infection of the 7th and 8th and the 8th, 9th, and 10th cranial nerves. The authors also mentioned a case of bilateral herpes oticus with total loss of hearing on the right side and moderate loss of hearing on the left.

HERPES OCCIPITOCOLLARIS

Herpes occipitocollaris signifies a herpetic infection located in the second and third cervical spinal ganglia; the cutaneous area of distribution is via the greater and smaller occipital, the great auricular, the cutaneous cervical, and the supraclavicular nerves. It may be noted that communicating branches with the facial and vagus nerves are present.

Motor paralysis complicating herpes zoster is not infrequent. The oculomotor nerves are said to be involved in 7% of the cases of Gasserian herpes zoster. Facial nerve paralysis is often seen in geniculate herpes zoster and occasionally in cases of Gasserian and occipitocollar herpes zoster. Paralysis of the soft palate, pharyngeal constrictors, and the intrinsic laryngeal muscles have been recorded in cases of vagoglossopharyngeal herpes zoster.

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9. McGovern, F. H.: South. M. J. **44**:137-140 (Feb.) 1951. Bateman, G. H., and Mawson, S. R.: J. Laryng. & Otol. **64**:17-18 (Jan.) 1950. Draffin, D. A.: Ibid. **64**:717-719 (Nov.) 1950.

10. Moffett, D. B., and McFarland, J. J.: Laryngoscope **59**:69-74 (Jan.) 1949.

11. McKenzie, D.: J. Laryng. & Otol. **30**:339-344, 1915.

12. Negus, V. E., and Crabtree, N. C.: J. Laryng. & Otol. **58**:192-195 (May) 1943.

13. Engström, H., and Wohlfart, G.: Herpes Zoster of Seventh, Eighth, Ninth and Tenth Cranial Nerves, Arch. Neurol. & Psychiat. **62**:638-652 (Nov.) 1949.

CONCLUSION

A summary is given of a concept of herpes zoster of the cranial nerves based on the pioneer work of Hunt and the more recent contributions by others, such as Tschiassny. As continued interest is focused on this subject, it appears likely that future clinical and histological studies will clear up many of the controversial and unknown factors.

ABSTRACT OF DISCUSSION

Dr. Kurt Tschiassny, Cincinnati: The authors presented an instructive classification of cephalic herpes zoster in terms of neural structures responsible for the distribution of dermomucosal eruptions and potentially associated sensory, secretory, and motor dysfunctions. Due credit is given to Hunt for his fundamental discovery of the geniculate ganglionitis responsible for the particular type of auricular herpes located in the "geniculate" zone. Hunt's ingenious application of the herpes zoster method for anatomical studies contributed fundamentally to our knowledge of the nerve supply of the external ear. It seems, however, that he accepted a somewhat totalitarian concept of the matter by postulating for all types of cephalic herpes associated with facial paralysis the simultaneous involvement of the geniculate ganglion. His error was revealed by the pathological observations of Denny-Brown and associates [4] and by my topognostic studies in 1946.[5] Hunt's attitude in this matter might be the result of a sort of "Pygmalion complex," Hunt's Galatea figure being symbolized by his self-created concept of geniculate ganglionitis. I must apologize for deviating into the field of psychiatry without a license to do so.

Herpes zoster represents in a certain sense a natural experiment of paramount exactness and selectivity. It offers numerous possibilities for the student of functional and descriptive anatomy. In a case of geniculate herpes which I previously reported, a unilateral ozena developed, resulting obviously from damage of the preganglionic secretory fibers for the nasal mucosa. Zarniko's theory regarding ozena as a neurodystrophic condition appears to be supported by this observation. Careful studies of the mobility of the soft palate in instances of isolated geniculate herpes and of the taste sensation in glossopharyngeal herpes may provide the answers to the still debated questions of the motor nerve supply to the levator veli palatini muscle and the supragenicular pathway of the chorda tympani taste fibers, respectively. There is nothing in this excellent paper with which I can take exception save one small point. It concerns the credit paid me by the authors for the method of topognosis in instances of facial nerve lesions which I described several years ago. I wish to emphasize again, as I have before, that my contribution in this matter represents merely a modernization and simplification of the pioneer work of Wilhelm Erb on the subject. Although I disagree in this small matter with the authors, I appreciate their friendly intentions. The presentation of Dr. McGovern and Dr. Fitz-Hugh is both informative and stimulating.

MANAGEMENT OF RECENT NASAL FRACTURES

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ONE CANNOT do a number of rhinoplasties without being forced to question the soundness of the management of recent nasal fractures. We believe that many difficult nasal reconstructions, tedious submucous resections, and extensive scar excisions could have been avoided had the proper treatment been instituted at the time of injury. Since the main respiratory current passes just beneath the dorsal arch,[1] and the nasal skeleton is practically subcutaneous,[2] even slight malalinements resulting from inaccuracies in reduction, such as unlocked fragments of bone, displaced spicules, or overriding bone edges—considered negligible in most fractures —may here cause serious impairment of function and conspicuous deformity.[3]

Through sad experience we have learned that to attempt a correction by blind reduction, using force in reverse of that which caused the fracture, often serves merely to aggravate the damage.[4] For instance, in fractures with lateral deviations, force applied to the side of the nose in an effort to push it back into the sagittal plane may cause overriding of the fragments,[4a, b] while in depressed fractures the introduction of a blunt instrument to elevate the bones often causes fragmentation of the contiguous bone margins (Fig. 1). Blind realinement of the fragments by unlocking and disimpaction[5] (as described later), while more effective, still lacks the assurance of precise reduction.

While we do not wish to deny that closed reduction for nasal fractures may give some measure of success in a good percentage of cases, nevertheless the many

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2. Schaeffer, J. P.: Nose, Paranasal Sinuses, Nasolacrimal Passageways, and Olfactory Organ in Man, Philadelphia, P. Blakiston's Sons & Co., 1920.

3. Fomon, S.: The Treatment of Old Unreduced Nasal Fractures, Ann. Surg. **104**:107 (July) 1936.

4. (*a*) Fomon, S.: The Surgery of Injury and Plastic Repair, Baltimore, Williams & Wilkins Company, 1939. (*b*) Watson-Williams, E.: The Broken Nose, Brit. M. J. **2**:791 (Oct. 31) 1931. (*c*) The Nose, Naso-Pharynx and Nasal Sinuses, in Bailey, H.: Emergency Surgery, Ed. 5, Baltimore, Williams & Wilkins Company, 1944.

5. Gillies, H. D., and Kilner, T. P.: Treatment of the Broken Nose, Lancet **1**:147 (June 19) 1929.

failures, the potential dangers incurred, and the uncertainty as to the restoration of alinement have left us with a feeling of dissatisfaction well expressed by Gross [6] in his epoch-making "System of Surgery" in reference to fractures in general:

> There is no class of injuries which a practitioner approaches with more doubt and misgiving than fractures. . . . If I were called upon to testify what branch of surgery I regarded as the most trying and difficult to practice successfully and creditably, I should unhesitatingly assert that it was that which relates to the present subject.

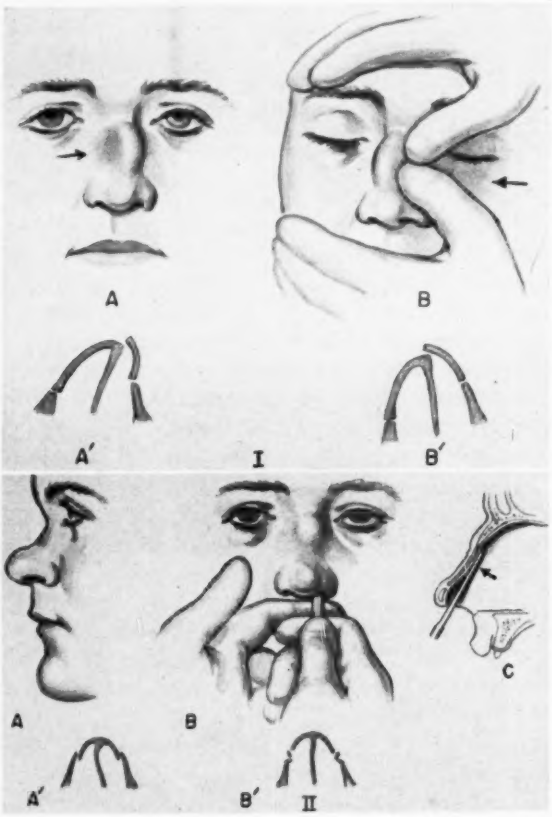

Fig. 1.—*I*, reduction of fracture by application of force in reverse of that which caused it. *A*, trauma applied to right side, with displacement to left. *A′*, diagram showing relationship of fragments. *B*, reduction by application of force to left side. *B′*, diagram showing resultant overlapping of fragments. *II*, reduction of fracture by application of force in reverse of that which caused it. *A*, trauma applied from in front, with saddling of dorsum. *A′*, diagram showing relationship of fragments. *B* and *C*, reduction by application of force from below. *B′*, diagram showing fragmentation of contiguous bone margins.

Our dissatisfaction with closed reduction prompted us to review the literature, in the hope that some of the advances in the treatment of fractures of the long bones might be applied with benefit to nasal fractures. Our interest centered on

6. Gross, S. D.: A System of Surgery, Philadelphia, Blanchard & Lea, 1859.

the work of that most meticulous of surgeons, Sir Arbuthnot Lane. On Jan. 8, 1894, he performed for the first time in Britain an open reduction for a fracture of a long bone.[7] A study of his fine book on "Operative Treatment of Fractures," [8] which summarizes 13 years' work and stands as a monument to that period of surgery, seemed to offer a promising approach to the solution of our problem. One can but wonder why this remarkable book had passed unnoticed by the rhinologist, for the applicability of open reduction to nasal fractures, through the rhinoplastic approach, seemed obvious. Perhaps this neglect on his part can be attributed to the intense preoccupation with his specialty. Gordon New,[9] in his presidential address before the American Laryngological Association, remarked: "The individual who does not keep abreast of the trends and discoveries in all fields of medicine but who confines his mental activities to the narrowness and limitations of his own specialty seldom conceives anything new."

Proving the applicability of open reduction to nasal fractures, however, came slowly and in stages. It took repeated conferences to reorient our thinking, many experiments on cadavers to adjust the technique, and numerous clinical trials to test the efficacy of the method. At first and with considerable trepidation we used it only when closed reduction failed, when the fragments could not be maintained in their corrected positions, and in cases with marked cartilage damage. We found the results so satisfactory that with the advent of antibiotics we extended its use and are now employing open reduction for practically all nasal fractures, limiting the closed method to simple fractures of the osseous vault.

Sufficient time has now elapsed to allow a summary of the advantages of open reduction. It permits (1) an accurate diagnosis by inspection, (2) precise reduction under direct vision, (3) evacuation of effusions and better drainage, (4) correction of preexisting defects, and (5) attention to associated fractures, and it lessens the danger of postoperative deformities and impairment of function.

We do not suggest that all fractures of the nose be treated by open reduction. For surgeons trained in rhinoplastic technique and with adequate facilities the open method offers the best results, but for those who are unable to avail themselves of the operative method closed reduction is safer and more serviceable.

We are still undecided as to the most appropriate method for treating nasal fractures in children. At present we are using closed reduction, not because it gives superior results but because we are reluctant to put the child through the hazards of a prolonged anesthesia. Nor have we the necessary instrumentarium. One of us, however, associated with a large children's service, plans to make the necessary modifications in the rhinoplastic instruments and to adjust the technique to the smaller anatomy. As soon as enough experience has been accumulated to justify a conclusion as to the comparative value of the open and the closed operations in children, we will publish the findings.

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7. Lane, W. A.: A Method of Treating Simple Oblique Fractures of the Tibia and Fibula More Efficient Than Those in Common Use, Tr. Clin. Soc. London **27**:167, 1894.

8. Lane, W. A.: Operative Treatment of Fractures, Ed. 2, London, Medical Publishing Company, Ltd., 1914.

9. New, G. B.: President's Address on Medical Progress, Ann. Otol. Rhin & Laryng. **60**:383 (June) 1951.

MANAGEMENT

While the treatment of nasal fractures must frequently include that of the contiguous facial bones, space will permit the discussion only of fractures limited to the nasal compound.

First-Aid Treatment.—The first-aid treatment common to all traumatic injuries applies here. It includes measures for the immediate preservation of life, such as the control of shock and hemorrhage, protection of the wound against further injury, relief of pain, and prophylactic inoculation. Only a few points will be mentioned.

Nasal fractures should be considered emergenices and quickly brought to surgical attention, for a delay may change a contaminated wound capable of being cleansed and united by a primary suture into an infected one that will heal slowly, with extensive scarring and deformity, and transform an easily reducible fracture into one difficult or impossible to reduce.

Shock takes precedence over all other considerations and is combated with the usual measures, such as elevation of the foot of the bed, rest, application of external heat, and replacement of blood volume by plasma, whole blood, fluids, and blood substitutes, as indicated. Oxygen is administered if necessary. If conditions warrant, a blood count is made and the blood typed for transfusion. Hemorrhage, as a rule, is not marked and will stop spontaneously. Bleeding parts are covered with gauze held in place with adhesive tape. If the dressing becomes soaked, it is overlaid with a similar one, only slight pressure being applied. Pain is relieved with morphine or meperidine hydrochloride (demerol®) in sufficient dosage to make the patient comfortable.

If the soft tissues have been damaged, no attempt is made to recover foreign bodies, for at the time of injury, aseptic conditions being absent, the manipulation would serve only to increase the contamination. The wound is "salted" with a sulfonamide powder and covered with a sterile dressing or, if this is unavailable, with a clean cloth wrung out of some antiseptic solution.

Antibiotics are given, such as 400,000 units of penicillin G procaine fortified; and if circumstances suggest soil contamination, 3,000 units of tetanus antitoxin, or a booster dose of 1 cc. of tetanus toxoid to patients already immunized. All medications are recorded on the chart.

When the patient has been placed in surroundings where proper facilities are available, definitive treatment is instituted at once.

Definitive Treatment.—Anesthesia: Topical and local nerve block is the method of choice, the same technique being employed as for a rhinoplasty.[10] If the patient is hypersensitive or is a child, a general anesthetic is administered.

Cleansing: The head is draped as for a rhinoplasty. If a wound is present, the first-aid dressing is removed and replaced with a fresh one. The tissues adjacent to the wound are scrubbed with septisol® or pHisoderm with hexachlorophene. Grease is removed with ether and blood with hydrogen peroxide. Foreign bodies embedded in the skin, such as bits of dirt, gravel, or clothing, are meticulously excised rather than dug out. The nasal fossae are cleansed, and all blood

10. Fomon, S., and Syracuse, V. R.: Routine Technic of a Typical Rhinoplasty, Illinois M. J. **94**:242 (Oct.) 1948.

clots and loose foreign bodies are removed by gentle suction and irrigation with warm saline solution. Hematomas under the mucosa, if present, are evacuated through an incision made at the lowermost part of the swelling. The dressing over the nasal wound is then removed, new gloves and gown are donned by the operator, the part redraped, and fresh sterile instruments provided. The wound is treated in the same manner as the surrounding skin. It is then surgically cleansed (débrided), i. e., devitalized, and contaminated tissue and foreign material are removed, to prevent infection and facilitate healing. (Chemotherapy is no substitute for débridement.) This procedure is carried out with discretion and a minimum of trauma. Dead tissue, such as loose tabs or corners and bits of crushed fat and subcutaneous tissue, are lifted out, but bone fragments, even when completely detached, are cleansed and replaced as grafts. Subcutaneous hematomas are evacuated. When there is doubt as to the viability of the skin, the decision to preserve or discard it will depend upon the amount which will be left for closure. If it is ample, the bruised edges are cut away in straight lines and without tailing,[4] so that they can be coapted at right angles. If not, every scrap is preserved, however slenderly attached for, although later some of it may be lost, ultimately there will be a greater saving than if all damaged skin had been removed at the outset. Even completely detached skin is cleansed, freed of subcutaneous tissue, and replaced as a full-thickness graft. Torn bits of the nasal tip or alae, even if completely severed, are carefully sutured back into place.

Closure: A decision must be made whether to close the wound or to leave it open, and this requires a careful and delicate appraisal of all the factors, such as the time which has elapsed since the accident, the degree of soft-tissue damage, and the amount of soiling.

Obviously, as long as the bacteria are still on the surface (contaminated wound), the wound may safely be closed by a primary suture after mechanical cleansing and surgical débridement. When the bacteria have invaded the tissues (infected wound), they cannot be reached by mechanical cleansing or surgical débridement, and excision would not only entail an unnecessary sacrifice of tissue but would also open up new avenues of infection and further decrease the viability of the part through the added trauma. Here the surgeon must content himself with delayed primary or secondary closure.

The determination of the exact time when contamination ends and infection begins constitutes the chief difficulty in the way of a decision as to appropriate treatment. As a rule the period of contamination lasts between 15 and 20 hours, but this is subject to a wide latitude. Infection is likely to occur early in patients whose resistance is already low; in the presence of exposure, hemorrhage, or shock; in the event of extensive local damage, as in lacerated, contused, and punctured wounds, and in cases of gross contamination, as in wounds inflicted out of doors, especially when the skin and clothing were dirty. In such circumstances infection may begin as early as two or three hours following injury. On the other hand, in cases in which the organisms are few and the pabulum scanty in tissues otherwise healthy, and in cases in which antibiotics have been used, infection may never actually establish a foothold.

1. Contaminated wounds (usually coming under observation within the first 20 hours): It is tremendously important that these wounds be closed by a primary suture, not only to prevent infection, but also to expedite bone healing.

If enough tissue remains to permit direct approximation, the parts are coapted. Every effort is made to minimize scarring by restoring the parts as nearly as possible to their former relationships. A V-tear is sutured as a V and a Y-tear as a Y,[11] to prevent the distortion which would otherwise result from the attempt to favor one dimension at the expense of another. Displaced flaps are returned to their original sites. Trap-door or slicing wounds require special care, as the tendency of the circular base to contract gives a bad ultimate scar.[12] Figure 2 is self-explanatory. Suturing of the wound is begun from essential landmarks, such as the inner canthus, lip, or naral border. In the absence of such points, gentle traction is exerted at the angles of the wound with dural hooks. With the wound edges thus reduced to a straight line, a start is made in the center of the wound and the remaining segments are bisected with each succeeding suture until the margins are coapted. The skin edges are brought together without vertical or horizontal deviation and without inversion. This is accomplished by incorporating an equal amount of symmetrically placed tissue in each bite of the needle. Figure 3 shows the direction of the sutures in irregular wounds. In the tying, tension is reduced to a minimum. Any wrinkling or discoloration caused by a suture warrants its immediate removal.

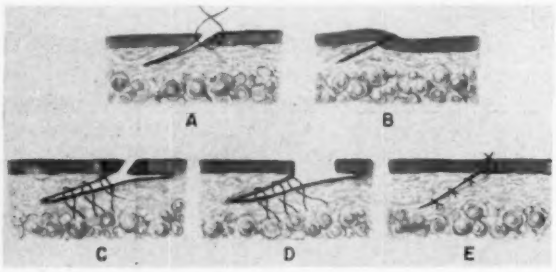

Fig. 2.—Management of slicing wound. *A*, slicing wound with trap-door flap closed in a single plane. *B*, resultant buckled scar, due to unequal contraction of wound base. *C*, skin separated from subcutaneous tissue; striped areas indicate thin edges to be pared. *D*, sutures passed, to close subcutaneous wound. *E*, subcutaneous and skin wounds closed separately and staggered, to break continuity of scar.

If there is not enough tissue remaining to cover the wound, rather than resort to undermining and stretching of the surrounding parts, it is best to cover the raw surface with a contiguous flap or a full-thickness graft, usually procured from the eyelid or from behind the ear. If a flap is used, the width of the pedicle should not be less than one-third the length of the flap.

The wound having been closed, it is covered with a layer of petrolatum gauze and a dressing is applied, with pressure not sufficient to constrict the blood vessels but enough to prevent postoperative edema, eliminate dead spaces, aid local splinting, and assist hemostasis. The dressing consists of sponge rubber, cotton waste, or fluff gauze held in place with elastoplast® or hooked adhesive strips connected with

11. Brown, B., and McDowell, F.: Plastic Surgery of the Nose, St. Louis, C. V. Mosby Company, 1951.

12. Maltz, M.: Surgical Treatment of Recent Facial Wounds, Eye, Ear, Nose & Throat Month. **23**:60 (Feb.) 1944; J. Internat. Coll. Surgeons **5**:334 (July-Aug.) 1942.

1-mm. rubber bands stretched twice their length.[13] It is not disturbed until the time for the removal of the sutures. Every other suture is cut on the third day and removed on the fourth, and the rest are removed on the following day.

2. Moderately infected wounds (usually seen within 20 to 40 hours—antibiotics have extended this period to as long as 10 days): These can frequently be closed by a delayed primary suture. The wound is cleansed and débrided and left open. If at the end of two or three days the wound is relatively clean, and the general condition indicates the absence of infection, it is closed without bacteriological control.

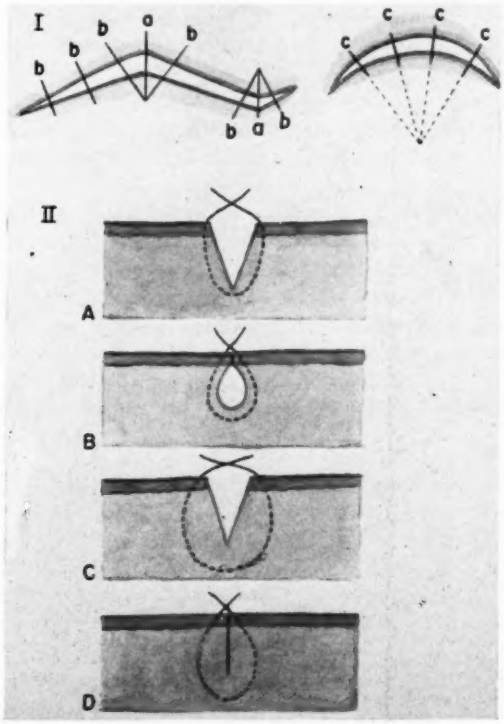

Fig. 3.—Method of suturing irregular wound. *I*, direction of sutures: *a*, suture made to bisect angle; *b*, limbs sutured at right angles to wound margin; *c*, in curved wound, shaft of needle taking direction represented by radius of circle, of which wound margins form circumference. *II*, sectional view. *A*, suture introduced parallel to wound surface. *B*, formation of dead space under skin, due to purse-string action of suture. *C*, correct manner of passing suture, which is carried well into tissues on each side. *D*, when suture is tied, whole depth of wound is brought together.

Delayed primary suture still permits the benefits of early healing and minimizes the danger of reinfection from the exterior. The cosmetic results nearly equal those following primary suture, although the approximation will be rendered somewhat more difficult, owing to infiltration of the tissues.

13. Overholt, R. H.: A Method of Securing Gauze Dressings on Draining Wounds, S. Clin. North America **12**:761 (June) 1932.

3. Definitely infected wounds (usually coming under observation after 40 hours): Closure is delayed until all signs of infection have been eliminated, for otherwise the inflammatory exudate under tension may spread the infection and/or press upon adjacent parts, interfere with the blood supply, and lead to strangulation of the tissues.

The course and treatment of the infection are governed in large measures by the character of the infecting organism. Therefore, cultures are made.[14] The material is obtained with cotton applicators. In addition, fragments of devitalized tissue are implanted in an appropriate medium for the purpose of detecting penicillin-resistant and anaerobic bacteria. The rationale of management is the same as that for infected wounds elsewhere: 1. To rid the wound of purulent exudate and necrotic tissue. The wound is washed with a sulfonamide or penicillin solution, and necrotic tissue and foreign bodies are removed, but the infected surface is not excised. Drainage is secured by application of warm, moist boric acid compresses renewed every three hours. 2. To limit bacterial growth by the use of antibiotics. The daily dosage of penicillin is 300,000 units. In severe infections 100,000 units of soluble penicillin is given intramuscularly every two or three hours, until the process is under control. If the organisms are penicillin-resistant or Gram-negative, or if there is a sensitivity to penicillin, other antibiotics, such as aureomycin, terramycin, streptomycin, and chloramphenicol (chloromycetin®), are employed. 3. To intensify the defensive reaction of the body by general supportive measures. As soon as the wound is comparatively clean, it is allowed to heal by granulation, closed by a secondary suture after the margins of the wound and the granulations have been excised, or covered with a thin razor graft, which is removed later and replaced with a full-thickness graft.

SKELETON

All are agreed that early reduction is essential to optimal healing and that fractures seen before the onset of swelling should be reduced without delay. Hippocrates,[15] in his third book, on the fundamental principles in the treatment of fractures, cautions that reduction is not to be delayed to the third day but should be carried out on the first day. Cave [16] writes in an editorial in *Surgery, Gynecology and Obstetrics*:

> The healing process of a fracture begins as soon as the bone is broken. This is well illustrated by the motion picture produced by the American College of Surgeons Committee on Fractures and Other Traumas. Any delay in treatment postpones healing. Therefore the bones should be realined as soon as possible. The hematoma about the fracture site serves a very definite function and the less it is disturbed the better.

The difference of opinion arises in cases when swelling has already taken place. There are those [17] who believe that immediate reduction is so essential to good union

14. Key, J. A.: Treatment of Compound Fractures in This Antibiotic Age, J. A. M. A. **146**:1091 (July 21) 1951.

15. Magnuson, P. B.: Fundamentals Versus Gadgets in the Treatment of Fractures, Surg., Gynec. & Obst. **62**:276 (Feb. 15) 1936.

16. Cave, E. F.: Fractures of the Long Bones, Surg., Gynec. & Obst. **89**:643 (Nov.) 1949.

17. Padgett, E. C., and Stephenson, K. L.; Plastic and Reconstructive Surgery, Springfield, Ill., Charles C Thomas, Publisher, 1948. Kazanjian, V. H., and Converse, J. M.: The Surgical Treatment of Facial Injuries, Baltimore, Williams & Wilkins Company, 1949.

that they set the fracture even if the contour of the skeleton is obscured by swelling, on the grounds that any interference with calcification causes a cessation of the process. The regenerative products must be eliminated before healing is resumed, and the longer the reduction is delayed the greater will be the amount of material to be absorbed. Thus the tissue may find itself without sufficient energy to take up the products and at the same time knit the bone.

Others [18] prefer to wait until the swelling has subsided (four to seven days). They argue that the swelling interferes with accurate palpation and coaptation of the parts, and that manipulation of the edematous and inflamed tissue may do more to delay healing or promote infection than the postponement of the management.

With due respect to the differences of opinion, for us the problem has been eliminated. Use of the rhinoplastic open method and of the antibiotics permits immediate reduction, regardless of the state of the overlying soft parts. Whereas formerly in cases of recent fracture we waited for the subsidence of the swelling and in those seen after consolidation (three weeks) we waited six weeks to three months to avoid the danger of latent infection, we now reduce the fractures when seen.

Fractures of the nasal pyramid are treated in the same manner as those of long bones, with these differences:

1. Long bones are fractured as a result of bending, and the displacement is due to muscular contraction. The relationship of the fragments to one another can be definitely established by palpation and roentgen examination, and reduction is accomplished by the application of skeletal traction to overcome the muscular pull. In the nasal bones the fracture is due to a compressive force acting on an arch-like structure, resulting in multiple fractures, the displacement being determined by the direction of the force. Here the malalinement cannot be accurately determined either by palpation or by roentgen study, and reduction is accomplished by disimpaction rather than by traction.

2. In fractures of long bones the parts require immobilization after reduction, to counteract muscular pull. In nasal fractures splinting is unnecessary for maintenance of reduction, since there are no muscles capable of causing displacement of the fragments and the bones are anatomically splinted by being "sandwiched" between two membranes.[19]

3. The mechanical fixation of fractured long bones in open reduction may cause pain, irritation, or suppuration and delay tissue repair. In nasal fractures this objection does not apply.

Osseous Vault.—Closed Reduction: This blind method of realinement can be carried out rapidly, but, as previously mentioned, it has a great disadvantage in that it leaves one in doubt as to the precision of the reduction. We limit its use to simple fractures involving the osseous vault, provided the upper and lower lateral cartilages have not been damaged.

Reduction is best accomplished by means of Walsham forceps (Fig. 4), one for the right and one for the left nasal wall and another for the septum. These instru-

18. May, H.: Reconstructive and Reparative Surgery, Philadelphia, F. A. Davis Company, 1947. Pick, J. F.: Surgery of Repair, Philadelphia, J. B. Lippincott Company, 1949, Vol. 2.

19. Hersh, J. H.: Management of Fractures of Nasal Bony Vault, Ann. Otol. Rhin. & Laryng. **54**:534 (Sept.) 1945.

ments are so designed that the blades fall short of meeting, thus eliminating the danger of damage by crushing. In the right and left forceps the inner blade is narrow and shaped to fit the inner surface of the nasal wall, while the outer blade is broad and made to correspond to the outer surface of the nasal wall. The septal

Fig. 4.—Walsham forceps for reduction of nasal fractures.

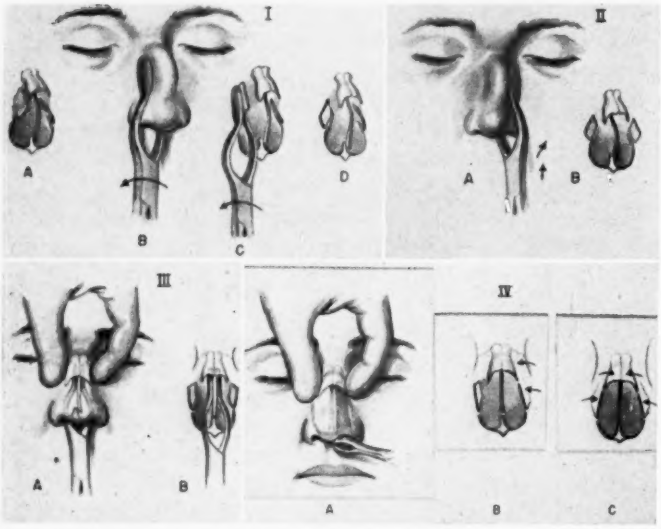

Fig. 5.—*I*, closed reduction of nasal fracture by disimpaction. *A*, diagrammatic representation of fracture, with displacement of fragments to left. *B*, fragments on broad side disimpacted and rotated outward with forceps. *C*, diagrammatic representation of *B*. *D*, diagram, showing completion of disimpaction. *II*, closed reduction. *A*, narrow convex side disimpacted by elevation and rotation outward. *B*, diagrammatic representation of disimpacted fragments. *III*, closed reduction. *A*, septum grasped with forceps and raised with upward and side-to-side motion. *B*, diagrammatic representation. *IV*, realinement of fragments. *A*, Killian speculum introduced into nasal fossa, to serve as "ironing board." Fingers, acting as "iron," press left fragments into place. *B*, diagram, showing fragments on left side reduced. *C*, fragments on right side reduced.

forceps has two broad flat blades and a curvature that permits grasping the septum without injury to the columella.

Technique: Obviously, if the fracture lines can be definitely delineated, there is no problem. For instance, a depressed fracture of one nasal wall can be reduced merely by grasping the nasal wall between the blades of the appropriate Walsham forceps and "ironing out" the displaced fragments by closing the blades. But usually the detailed alinement of the displaced fragments cannot be definitely diagnosed, either by roentgenogram or by palpation, and, irrespective of the type of fracture, an over-all reduction is necessary to insure the restoration of the integrity of the arch. The fragments, impacted or locked in faulty relationships, must be mobilized and molded into proper position.[5]

The technique is as follows:

In fractures with lateral displacement the broad flat side is grasped with the forceps, and with an outward movement of the wrist the nasal wall is gently rotated outward, until the bones are disimpacted (Fig. 5 *I*). The narrow wall is grasped with the forceps in like manner. The bones on this side, being not only impacted but overriding, are first lifted and then rotated outward, as on the opposite side (Fig. 5 *II*). The various lines of fractures can now be easily and accurately outlined by palpation. The septum is then grasped with the Walsham septal forceps and by a gradual upward and side-to-side movement is made to assume a central position (Fig. 5 *III*). If the force came from in front, the procedure of elevation and outward rotation is carried out on both nasal walls and the septum is brought into the sagittal plane, as before. The disimpacted fragments are now "ironed out." A long Killian speculum is introduced into one nasal fossa and opened, to serve as an "ironing board," and the fingers, acting as the "iron," press the fragments into place. The procedure is repeated on the opposite side (Fig. 5 *IV*). Finally, the Walsham forceps is made to grasp the septum, and the assembled fragments are checked with the fingers for the detection of any inaccuracies in reduction. Intelligent use of force is required. Undue violence is likely to cause additional fractures and/or overriding of the fragments. If the parts do not fall into easy alinement, if the fragments can be held in position only with the aid of splints, or if the cartilages have suffered considerable damage, it is better to do an open reduction, although the result will not be as good as if this procedure had been employed initially.

The admonition of Sir Robert Jones [20] in relation to long bones applies equally well in nasal fractures:

The lesson is therefore clear that whatever methods be adopted they must be efficient from the first. If a surgeon is doubtful whether he can treat a fracture efficiently by non-operative means, he ought to consider whether he cannot do better by operating at once. He ought not to say, "Well, we can see what becomes of it, and if it is not satisfactory we can operate later," for by so doing the opportunity of getting a good functional result may be irretrievably lost.

Cartilaginous Vault.—After reduction of the bones, the cartilaginous skeleton is attended to. Because of the firm attachment of the cartilages to the bone, the osseous reduction will nearly always bring displacements of the upper part of the cartilages into proper position. The distal margins will require some additional manipulation and molding, to pull them into place. If the septum is fractured, the upper fragment may be pried into position and maintained by a suture, as suggested by Peer.[21] If it has slipped out of the septal groove, it is rocked from side

20. Jones, R.: Presidential Address on the Present Position of the Treatment of Fractures, Brit. M. J. **2**:1589 (Dec. 7) 1912.

21. Peer, L. A.: An Operation to Repair Lateral Displacement of the Lower Border of the Septal Cartilage, Arch. Otolaryng. **25**:475 (April) 1937.

to side, until it clears the maxillary spine and finds its normal place. Should this maneuver fail, reduction can usually be brought about by placing a MacKenty elevator beneath the displaced edge and prying it over the margin of the maxillary spine into the septal groove (Fig. 6). In any event, the cartilages are never left for a later reduction, for in the process of repair fibrosis will bind them down and make readjustment difficult or impossible.

If properly reduced, the fragments will maintain their corrected position without support. However, a light petrolatum pack is introduced into the nasal fossae and a Stent dressing applied, to offset any possible disturbance or accidental trauma on

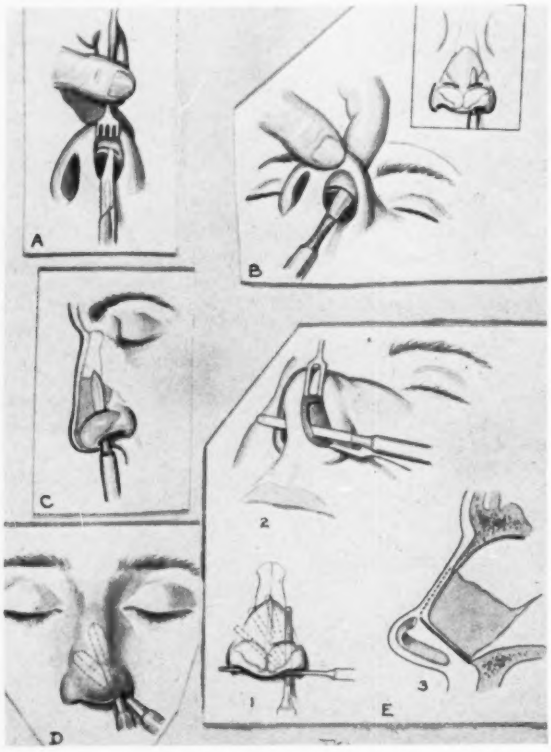

Fig. 6.—Open reduction. Uncovering of nasal pyramid. *A*, plica nasi exposed and aponeurosis connecting upper and lower lateral cartilages incised with Bard-Parker No. 11 knife. *B*, soft structures overlying upper cartilaginous vault elevated; double-edged knife introduced into vestibular incision and carried to caudal margin of nasal bone in plane above perichondrium. Insert, ventral view. *C*, knife swept from side to side, separating soft structures. *D*, separation of soft structures continued over dorsum with blunt, curved, double-edged knife. *E*, caudal exposure of nasal pyramid: *1*, button-end knife introduced, advanced to nasofrontal suture and swept obliquely downward, until tip appears through opposite intercartilaginous incision; *2*, membranous septum cut through; *3*, line of separation.

the part of the patient through violent sneezing or turning in bed, and also to prevent postoperative swelling. In our hands the use of complicated splints has proved unsatisfactory. If the bones have been correctly adjusted, splints are not necessary; if they were improperly reduced, splints which forcibly hold them in

place will do more harm than good. The innumerable prosthetic devices described for this purpose are *ipso facto* evidence of their failure.

Mucosal Lining.—Damage to the nasal lining is treated in accordance with the same principles as those applying to the skin. The airway is meticulously inspected, lacerations are sutured, hematomas evacuated, and all raw surfaces skin-grafted, to prevent the formation of synechiae.

Open Rhinoplastic Reduction: We have evolved the following procedures, which up to this time have given us better results than those of the past.

Technique: The technique of an open reduction is the same as that of the rhinoplastic operation and is amply described in the literature.[4a,b] Only passing mention will be made of the steps. 1. The nose is aseptically prepared and anesthetized. 2. With a No. 11 Bard-Parker knife an incision is made in the aponeurosis between the upper and lower lateral cartilages (Fig. 7*I*). The procedure is repeated on the opposite side. 3. A double-edged Joseph knife is introduced through the incision, and the soft tissues over the sides of the pyramid are elevated. 4. With a blunt double-edged Fomon knife the soft structures over the dorsum are elevated. 5. A button-

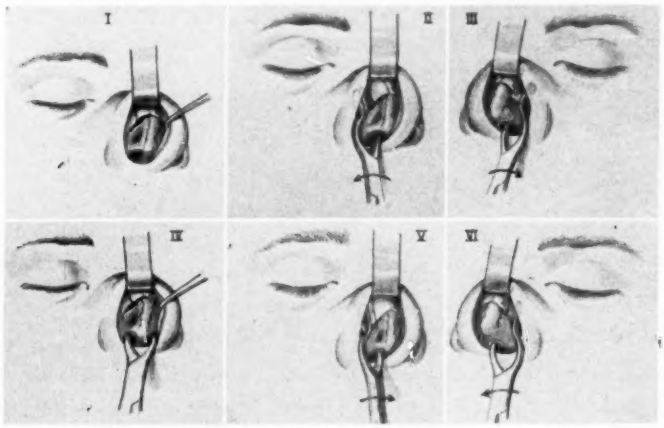

Fig. 7.—*I*, soft tissues elevated with angulated retractor, and pyramid inspected, to show lines, directions and displacements of fragments. *II*, broad blade of Walsham forceps placed on outer surface of nasal wall beneath skin. Narrow inner blade placed on mucosal side, and fragments disimpacted by rotation of blade outward. See Figure 5*I*. *III*, short side of pyramid lifted and rotated outward. See Figure 5*II*. *IV*, septum grasped with forceps and raised with upward and side-to-side motion. See Figure 5*III*. *V*, reduction of fragments. Forceps manipulated to bring right nasal wall into alinement. See Figure 5*IV*. *VI*, reduction of fragments. Forceps manipulated to bring left nasal wall into alinement. See Figure 5*IV*.

end knife is introduced through the left incision, passed over the dorsum, and carried down until it emerges through the opposite intercartilaginous incision, at which point it is turned at right angles and made to follow the caudal extremity of the septum, cutting through the membranous septum down to the nasal spine. 6. An angulated retractor is introduced, to elevate the overlying soft parts, and the pyramid is inspected, to show the lines, directions, and displacements of the fracture (Fig. 7*I*). 7. The bony arch is then reduced with a Walsham forceps, the same technique being employed as in closed reduction, except that the outside blade is placed under instead of over the skin (Fig. 7*II*, *III*, *IV*, *V*, and *VI*). The result of the reduction is checked by inspection. 8. The upper lateral cartilages are unbuckled and realined. To facilitate their reduction, it may be necessary to cut them away from the septum and restore them to proper alinement. 9. The septum is treated as in a closed reduction. If there is any interference with realinement, one membrane is elevated (Fig. 8), and the displaced cartilage

is brought back into its normal position. If there is a tendency toward recurrence of the displacement, we excise the overlapping piece rather than suture the fragments. 10. After the reduction, the membrane is sutured back into place.

Diagnosis.—The appropriate treatment can be instituted only after a proper evaluation of the evidence obtained from (1) a general appraisal of the patient, (2) a history of the accident, and (3) a local analysis of the injury.

1. A general appraisal of the patient is made for the detection of possible damage to other parts, the care of which may be more urgent for the preservation of life than that of the nasal fracture—for example, a thoracic or abdominal wound or an ethmoid fracture.

2. A history of the accident. Knowledge of the size of the traumatizing agent, the site and the direction and degree of the force will aid in determining the extent of damage and help establish the type of fracture. A history of previous nasal fractures and deformities and a photograph of the patient taken prior to the accident will be useful not only to enable one to avoid mistaking a natural peculiarity for a fracture deformity but also to rule out the patient who seeks to exploit a recent fracture in order to force the correction of a preexisting deformity.

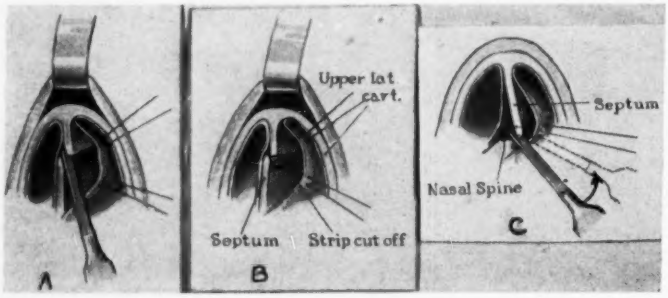

Fig. 8.—Reduction of septal fracture. *A*, membrane elevated on one side; cartilage pried into place. *B*, overlapping cartilage excised, to facilitate reduction. *C*, displaced edge of septal cartilage pried into septal groove with MacKenty elevator.

3. Local analysis of the injury must include the skin, osseous skeleton, and cartilaginous skeleton.

(*a*) Skin: The damage to the skin and the amount of skin available for closure can usually be determined by inspection, and the possibility of infection, by a history of the circumstances surrounding the accident.

(*b*) Osseous Skeleton: A history of a blow on the nose associated with nosebleed (more abundant in cartilaginous fractures), ecchymosis about the eyes, pain and swelling, and obstruction to breathing is presumptive of a nasal fracture. If, in addition, there is a marked flattening of the nasal arch, with a displacement of the tip upward and backward ("pug nose") or a deviation showing a broad concavity on one side and a narrow convexity on the other, the diagnosis can be made at a glance. But, unfortunately, inspection is of little value unless the patient is seen immediately after the injury, and even then it fails to give precise information as to the site of fracture, line of overriding, or deviation. Palpation is of more value.

If the swelling is not too marked, firm continued pressure with the tips of the fingers can often push the edema aside, so that the deeper structures may be palpated. Care is taken to avoid mistaking a hematoma or a preexisting asymmetry or flatness for a recent fracture. Crepitus should not be sought for too energetically and may be absent, for the fragments are splinted by swollen periosteum, coagulated blood, and edema.

X-ray study is of limited value. It reveals a definite fracture in only about 50% of cases.[22] There are as yet no definite positions in use which give a clear picture of the true nature of the fracture. The posteroanterior view may show only the presence of septal fractures. In a lateral view one side of the bony vault is superimposed on the other, obscuring the details. A frontomental view provides the most information. The roentgenogram fails to show the alinement of the fragments except in deviations of the lower end of the nasal bone. For children x-ray examination is of even less value because of incomplete ossification. Nevertheless, since nasal fractures often become a matter of litigation, it is advisable to secure roentgenograms of the injured part before, during, and after treatment.

(*c*) Cartilaginous Skeleton: After the area has been anesthetized and the mucosa shrunk, the nasal fossae are cleansed, the vibrissae cut, and the cavity meticulously explored, to ascertain the damage to the cartilages. Inspection may show a tear in the membrane, which indicates the site of the fracture, projecting bone, displacements, buckling, or fracture of the septum and the lateral cartilages. By means of a probe we determine the site of the fracture lines discovered on external examination.

While a definite diagnosis of nasal fracture is often impossible, nevertheless, in view of the marked impairment of function and gross deformity which frequently follow these traumatisms if neglected, we believe that it is sound practice in presumptive cases to treat the condition as though it were a fracture until proved otherwise.

Mechanism: A review of the surgical anatomy and a few experimental nasal fractures and their attempted reduction in the anatomic laboratory will do much to clarify the mechanism.

Nasal fractures are always due to direct violence. As a result, the fragments are usually locked or impacted. Because of the close adherence of the mucosa to the skeleton, they are almost invariably compound. The nasal pyramid is composed of many bones and cartilages, and it is seldom that a fracture is limited to any one segment. The varieties are innumerable and have been amply described in the literature. For us they are merely of academic interest, for we are rarely able to palpate them in detail, and frequently they escape detection in the roentgenogram.

A. Fracture of Bones. The character of the fracture is governed by (1) the architecture of the nasal pyramid, (2) the degree and direction of the force, and (3) the size and shape of the striking agent.

1. Architecture of nasal pyramid. The upper third of the nasal pyramid is a solid block of bone composed of the thickest part of the nasal bones, which articulate

22. Becker, O. J.: Problems of the Septum in Rhinoplastic Surgery, Arch. Otolaryng. **53**:622 (June) 1951. Becker, O. J.: Nasal Fractures: An Analysis of 100 Cases, Arch. Otolaryng. **48**:344 (Sept.) 1948.

in a "halving" union with the frontal bone, which, in turn, is underpinioned by the nasal spine of this bone. This part of the pyramid serves as a fixed buttress to unite the facial with the cranial skeleton. It is so strongly constructed that great force is required to fracture it. The lower two-thirds of the pyramid is constructed on the plan of an arch (Fig. 9). The nasal bones constitute the keystone. They abut each other in a juxtaposition joint, the two bones lying in immediate contact, devoid of articular surfaces or ligaments, like the gable of a roof. They articulate with the frontal processes of the maxillae by a suture, like a cogwheel-and-ratchet device, reciprocally immovable. The frontal processes of the maxillae form the voussoirs (Fig. 9), the walls of the maxillary sinuses the abutments, the margins of the piriform processes the imposts, and the maxillae the piers. This arch-like construction provides a strong protection against external force.

2. Degree and direction of force. Slight trauma will cause chip fractures, which often tear through the mucosa and project into the nasal cavity, or fissured fractures, longitudinal or transverse in direction. Greater force will result in comminution and/or impaction.

If the blow was from in front, considerable violence will be required to fracture the upper part of the pyramid, whereas the lower, more fragile portion will yield

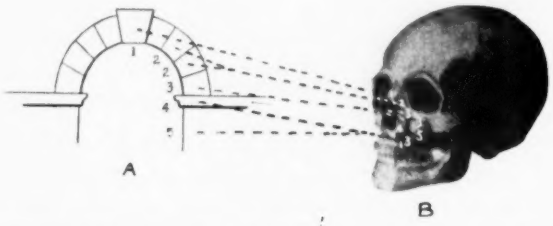

Fig. 9.—Comparative architecture of nasal pyramid. *A*, diagram of arch: *1*, keystone; *2*, voussoir—side wall of arch; *3*, abutment—supporting structure, to sustain lateral pressure; *4*, impost—place upon which arch rests; *5*, pier—support of arch. *B*, structure of nasal pyramid: *1*, nasal bones—keystone; *2*, frontal process of maxilla—voussoir; *3*, walls of maxillary sinus—abutment; *4*, margins of piriform opening—impost; *5*, maxillo-canine-infraorbital buttress—pier. Dotted lines indicate corresponding parts.

to a lesser force. A blow which bears down directly on the key of the arch (the two nasal bones behaving like a single bone) will act on the dorsum as it would upon an arch which, according to a law of mechanics,[23] must fracture into three or four pieces (Fig. 10), producing (1) bilateral fracture of the nasomaxillary gable, with or without displacement, depending on the obliquity of the force; (2) unilateral fracture, usually without displacement; (3) telescoping of nasal bones between the frontal processes, with a widening and flattening of the dorsum, or (4) fracture through the frontal processes, with separation along the internasal articulation, the fragments under- or over-riding the maxillae like an open book laid face down (Fig. 10).

Lateral force is the most frequent cause of nasal fractures. At the point of impact the bones are fractured and separated from their osseous unions and are

23. Kidder, F. E.: Architects' and Builders' Handbook, Ed. 17, New York, John Wiley & Sons, 1921. Kleitz, A. S.: Personal communication to the authors.

carried as a whole in the direction continuing the line of the blow, so as to produce the typical broad concave surface on that side. The wall opposite the point of impact is buckled, or it overrides or underrides the maxilla, producing a short convex side (Fig. 5*I*).

When the force is from above, the protection afforded by the forehead to the upper part of the nose prevents many fractures in that area. It is the inferior, more fragile, portion which is subject to damage.

Force from below may cause overriding of the frontal bone, with a loss of the glabellar angle, or it may jam the nose into the base of the skull.

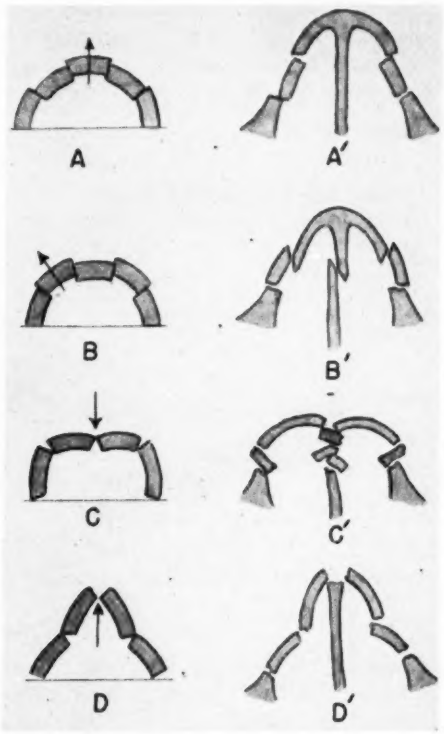

Fig. 10.—Mechanics of nasal fracture. *A*, failure of semicircular arch; haunches sliding down. *A′*, nasal fracture. *B*, failure of semicircular arch; haunches sliding up. *B′*, nasal fracture. *C*, failure of semicircular arch; opening of arch ring. *C′*, nasal fracture. *D*, failure of pointed arch; opening of arch ring. *D′*, nasal fracture. For details, see text.

3. Size and shape of striking agent. A small sharp instrument is likely to cause injury localized at the point of impact, whereas one with a broad surface will produce displacement, locking, and impaction of the fragments.

B. Fracture of Cartilages. Owing to their elasticity, the cartilages escape a great number of traumatisms. Damage to the cartilage, however, leads to more serious consequences than injury to the osseous skeleton. Reduction of the bones usually offers little difficulty, and physiologic disturbance is minimal, owing to

the location of the bones above the course of the respiratory current. Furthermore, any residual deformities can easily be corrected at a later date by a rhinoplasty. The cartilages, however, are more affected by scar contraction; organization of the inflammatory exudate leads to buckling and curling, and hematomas and chrondritis tend to undergo calcareous degeneration. These changes may render later correction difficult if not impossible.

The upper lateral cartilages and septum form the upper cartilaginous vault and act as a unit. In frontal force the upper lateral cartilages buckle, fracture, or are torn from their bony attachments. The septum, caught between the traumatizing agent and the fixed attachment to the vomer and ethmoid bone, is subject to damage depending upon two factors: (1) its attachment to the vomerine sulcus and (2) its relationship to the upper lateral cartilages. If the cartilage is firmly fixed to the vomerine sulcus, it will bend on fracture. If the sulcus is shallow, as in children, it will slip out of the articulation and be dislocated into one or the other nasal

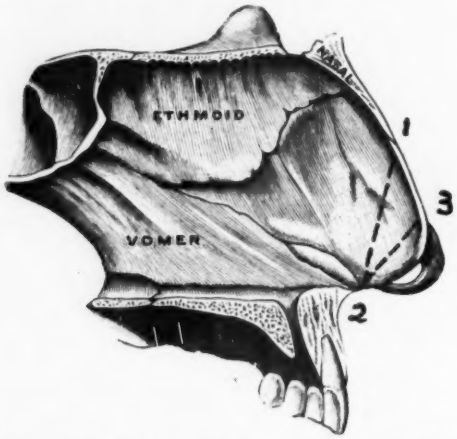

Fig. 11.—Effect of trauma on septal cartilage: 1—2, line of fracture when upper lateral cartilages do not extend full length of septal cartilage; 3—2, line of fracture when upper lateral cartilages extend full length of septal cartilage.

fossa. If the upper lateral cartilages do not extend the full length of the septum, a fracture will occur running from the caudal end of the nasal bone to the nasal spine of the maxilla.[24] If the upper laterals extend the full length of the septum, the fracture will follow a line from the caudal margin of the upper laterals to the ventral extremity of the vomerine sulcus (Fig. 11). The posterior (cephalocaudal) fragment, fixed in the ethmovomerine angle, is not displaced. The more movable anterior (ventrocaudal) fragment may rotate as much as 90 degrees, to lie transversely across the columella, so that its upper border is displaced into one fossa and its lower border projects into the other fossa. A slit in the membrane may permit the free end of the cartilage to project.

Bone, unlike soft tissue, does not heal by fibrous tissue, but the loss is replaced by a metaplasia of the anaplastic cells, with mineral depositions, forming a structure

24. Huet, P. C.: Treatment of Recent Nasal Fractures, Médecine **9**:317 (Jan.) 1928; Recent Nasal Fractures, J. chir. **31**:649 (May) 1928.

identical in character to the original bone. Since the bones of the nasal pyramid are membranous, healing takes place not by the preformation of cartilage but by the production of osteogenetic fibers, with a deposition of calcareous granules. Very often the fragments do not undergo bony union but remain joined by dense connective tissue which unites the periosteum with the endosteum. We have often been able to shift the nasal pyramid a year or two following an osteotomy fracture.

Unlike bone, fractured cartilage does not reunite by cartilage. The fragments are bound together by an ingrowth of connective tissue.[25]

Complications.—Without entering into the question of who should treat the associated fracture, we believe that the rhinologist, with his special knowledge of the anatomy of the head and with adequate training, is best qualified. A brief review of the anatomy is essential to a clear understanding of the mechanism by which complications are brought about.

The nasal cavity, enclosed in the maxillary compound, can be compared to a mezzanine floor between the cranial and oral cavities. Its shape approximates the figure of a recumbent three-sided prism (Fig. 12). The triangular ventral surface is occupied in large measure by the piriform opening, whereas the dorsal surface is made up of the choanae. The floor is rectangular, with a length of 65 mm. and a

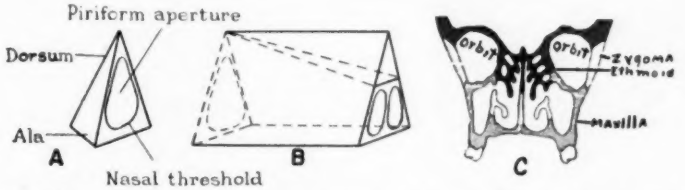

Fig. 12.—Diagrammatic representation of nasal cavity. *A*, external nasal pyramid. *B*, sagittal section of nasal fossa. *C*, coronal section.

width of 32 mm.[26] The lateral walls converge to meet in the midline about 45 mm. above the floor. The roof is arched, so that the height in the center is greater than that in front or behind. On a coronal section the roof is narrow (Fig. 12) and helps to form the floor of the anterior cranial fossa. The summit is composed of the cribriform plate and the lateral masses of the ethmoid bone. The sides comprise the lacrimal bone and the orbital plates of the ethmoid bone and maxilla. The floor, which constitutes the premaxilla, forms the roof of the oral cavity. In front of the nasal fossa proper is a triangular pyramid which encloses the external nasal cavity (vestibule) (Fig. 12). The apex is directed upward and attached to the frontal bone, and the base forms a triangle which outlines the nostrils. The nasal cavity throughout is divided by a septum into two parts, but only in the external nose is this partition cartilaginous.

Early complications are due mainly to extensions of the nasal fracture to contiguous parts—the olfactory area, frontal sinus, cranial cavity, ethmoid labyrinth,

25. Zuckerkandl, E.: Normal and Pathologic Anatomy of the Nasal Cavity, Vienna, W. Braumuller, 1882-1892.

26. Mink, P. J.: Physiology of the Upper Respiratory Passages, Leipzig, F. C. W. Vogel, 1920.

orbits, lacrimal canal, and maxillary sinus. The associated fracture may be unilateral or bilateral and is usually compound and impacted.

Because of the anatomic structure of the contiguous facial bones, the line of fracture is more or less constant,[27] generally taking a horizontal course. Vertical fractures are rare, since the more solid portion of the bone is encountered in this direction. Owing to the absence of strong muscular attachment, the displacement is principally in the line of continuation of the force, although gravity also plays a part. Figure 13 shows the sites of predilection.

If the traumatizing force is delivered from in front and to the upper part of the nasal pyramid, there usually results a transverse unilateral or bilateral fracture through the orbit, nasal fossa, and pterygoid and zygomatic processes, the extent of the injury varying from a fissure to a complete separation of the facial bones from the base of the skull. The whole mass of bone may sag down and produce an elongation of the face; or the entire central section, comprising the maxillary, nasal, and ethmoid bones, may be driven backward, the upper teeth becoming alined

Fig. 13.—Complicating nasal fractures. Frontal and lateral views, showing lines of weakness through (*1*) orbits; (*2*) middle of maxillary compound; (*3*) palatal process of maxilla; (*4*) frontomaxillopremolar region; (*5*) maxillocanine region.

behind the lower, so that the countenance assumes a dish-faced appearance. If the force is delivered lower down, the line of fracture passes either through the antrum and nasal fossa or through the alveolus and palate, the resultant damage ranging in severity from the splintering of an alveolus to an impaction or complete separation of the entire alveolar-palatal compound. If the force is directed from below, the maxillofacial compound will be carried upward toward the base of the skull; if from the side, the brunt of the blow will be received by the malar-zygomatic compound, which will be impacted into the antrum, the line of fracture extending radially to the maxilla, the infraorbital ridge, and the alveolar process.

Generally speaking, if the patient is seen early, before swelling and emphysema develop to obscure the physical findings, these fractures can be easily diagnosed

27. Le Fort, cited by Fomon, S.: The Surgery of Injury and Plastic Repair, Baltimore, Williams & Wilkins Company, 1939.

from a history of the injury, complaints of pain on mastication, malocclusion of the teeth, and hemorrhage from the conjunctiva, nose, and mouth. But in the presence of extensive soft-tissue damage or cerebral injury the condition may escape notice and be discovered only when the patient finds the malocclusion of the teeth interfering with mastication. Simultaneous palpation of the orbital borders, zygomatic arches, nasal and maxillary bones, and the two sides of the palate will disclose asymmetry and reveal the extent of the fracture. Localized tenderness referred to a constant point, especially when aggravated by lateral compression of the two sides of the face, is particularly significant. In the case of nonimpacted fractures crepitus may be elicited by gentle manipulation of the upper jaw with the fingers of the right hand while the temporal region is immobilized with the fingers of the left. Injury to the infraorbital nerve is evidenced by alteration in sensation over its area of distribution.

X-ray examination clearly reveals the site and extent of the fracture and the involvement of the dental roots. A fracture of the alveolar process will be shown up most effectively if a dental film is used; fractures extending into the orbit are best disclosed by a posteroanterior view and backward displacements by a coronomental view.

Late complications comprise deformities of the osseous vault due to impaction, overlapping of fragments, lateral displacement of the skeleton, and angulation. Deviations of the cartilaginous nasal bridge and distortion of the tip, due to cicatricial contraction of the soft tissues, are common. Since the periosteum is more adherent to the mucosa than to the skeleton, it separates from the bones and becomes displaced. Its osteogenetic activity will cause it to produce new osseous layers, which may show up as an exostosis. The peeling off of the mucosa may lead to laceration of the terminal branches of the olfactory nerve, with an impairment or loss of the sense of smell. Narrowing of the nasolacrimal canal may cause epiphora.

After-Treatment.—The after-treatment of nasal fractures is conspicuous by its absence. Unless complications arise, the less the parts are interfered with in the postoperative period the better. The patient should be instructed not to blow the nose and told that the swelling will take months to subside.

SUMMARY AND CONCLUSIONS

With the best treatment there are cases in which deformity and impairment of function cannot be prevented. In this relation we should like to paraphrase Sir Astley Cooper,[28] who in 1822 wrote the first modern treatise on the treatment of fractures. This book became so popular that by 1839 it had gone through 10 editions. His admonitions could very well apply to nasal fractures:

> These cases should teach the members of our profession to be kind, generous, and liberal toward each other, and not to impute to ignorance or inattention that which is the result of a generally irreparable accident. It too often happens that when every trial has been made to restore the parts and without success, the patient goes to some other surgeon to whom he shows the deformity and calls attention to the impairment of function. A jealous and illiberal medical man might say, "Yes, this is a fracture which has not been properly reduced. I wish I had seen it at the first, but now it is too late for a successful attempt to correct it." However,

28. Cooper, A. P.: Treatise on Dislocation and Fractures of the Joints, London, published by the author, 1822.

every intelligent well informed surgeon will now confess that no knowledge or exertion of skill on his part could have prevented the deformity and impairment of function which results from this . . . accident.

We have endeavored in this discussion to show the benefits of the open operation, in the hope of contributing something of value to the standardization of the treatment of recent nasal fractures.

A comparison between the closed and open reduction may be summarized as follows:

Closed Reduction	Open Reduction
Advantages	
Rapid coaptation of fragments is assured.	Diagnosis can be made on inspection.
No special instruments are required.	Precise realinement of fragments is assured.
Rhinoplastic training is unnecessary.	Danger of postoperative deformity and impairment of function is lessened.
Disadvantages	
Offers no aid to diagnosis.	Is time-consuming.
Gives no assurance of precise reduction.	Requires special instruments.
Results often in postoperative deformity and impairment of function.	Requires training in rhinoplasty.
Indication	
Limited to simple fractures of osseous vault	Applicable to all types of nasal fractures, especially with cartilage involvement.
Contraindicatisn	
Extensive cartilage damage	Lack of adequate training and equipment

RESPONSE OF OTOSCLEROSIS AND ADHESIVE OTITIS TO TREATMENT WITH CORTICOTROPIN

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COPENHAGEN, DENMARK

CORTICOTROPIN (ACTH) is a pituitary hormone with a stimulating action on the adrenal cortex. One of the functions of the adrenal cortex is regulation of the activity of mesenchymal tissue. In many patients with diseases of such tissue administration of corticotropin has had a favorable—sometimes almost dramatic—effect after a few days of treatment, especially if therapy was instituted during the first stage of the disease.

Corticotropin has a pronounced inhibitory effect on the increased mesenchymal activity which is a common feature of the diseases improved by treatment with this hormone. (Bjørneboe [1] recently presented a survey of the effects of corticotropin therapy.)

On the basis of clinical and experimental experiences with corticotropin from other fields, especially its effect on certain vascular and articular lesions, it seemed natural to investigate its effect on patients with impaired hearing owing to otosclerosis and adhesive otitis, these diseases being due to morbid processes in the mesenchymal tissue of the ear. Further studies should be made on the effect of corticotropin therapy in patients with recurrent otitis, in which allergic reactions may be a concurrent cause.

CLINICAL STUDY

In the Ear Department of Sundby Hospital we have so far treated 15 patients with corticotropin. Of these, 12 had otosclerosis and 2 adhesive otitis; 1 was treated after fenestration on account of aplasia of external and middle ear. Treatment of patients with recurrent otitis has not yet been carried out.

The effect of corticotropin was investigated under three conditions differing in principle: (1) in ears without operation, (2) in ears after fenestration when the improvement in hearing obtained after the operation had been lost again, and (3) in ears immediately following fenestration, when corticotropin was given as after-treatment.

All the patients were given a dose corresponding to 20 to 30 mg. (Armour standard) of corticotropin [2] intramuscularly four times daily for eight days. This is a short period of treatment but has so far been the only one possible, owing to difficulty in procuring the substance. The biologic action of the hormone was followed by daily counts of eosinophilic leucocytes in the blood. In all cases a marked effect of corticotropin was observed, i. e., a fall in esinophils to 0 or to very low values during the treatment. In addition, the patients were weighed daily to observe the fluid retention. Finally, electrocardiograms were made, and the hormone level (17-ketosteroids) was determined.

Read before the Danish Otolaryngological Society, May 19, 1951.

From the Ear Department, Sundby Hospital (Prof. Robert Lund, M.D.).

1. Bjørneboe, M.: Nord. med. **43**:822, 1950.

2. Corticotropin was supplied as "acton," by the Frederiksberg Chemiske Fabrikker A/S.

TABLE 1.—*Results of Corticotropin Treatment in Nine Otosclerosis Patients with Labyrinthine Fenestration Performed Nine to Thirty Months Previously*

Patient	Age, Yr.	Sex	Ear with Fenestration	Fistula Symptom		Average Loss of Hearing, Db. at 512, 1024, and 2048 Cps										Subjective Improvement
				After Operation	During ACTH Therapy	Before Fenestration		Minimum After Operation		Mo. Postoperatively	Before ACTH Therapy		Mo. Postoperatively	After ACTH Therapy		
						Air	Bone	Air	Bone		Air	Bone		Air	Bone	
1	28	M	Left	÷ *	÷	40/47	28/30	37/45	23/20	6	50/62	35/27	30	50/62	33/25	None
2	34	F	Left	÷	÷	37/38	3/6	32/43	2/10	½	50/60	30/42	16	50/62	25/35	None
3	40	M	Left	+	÷	60/73	28/25	57/35	33/30	12	70/53	48/40	37	68/57	45/40	None
4	43	M	Right	÷	÷	58/52	23/20	50/65	(25)/(25) †	6	58/65	38/35	19	62/58	48/37	None
5	44	M	Right	÷	÷	65/48	(22)/(20)	55/48	(28)/(22)	1½	53/65	40/36	30	60/62	42/47	None
6	48	F	Right	÷	÷	58/63	32/37	48/62	42/40	3	58/63	43/38	30	60/62	38/35	None
7	52	F	Right	÷	÷	65/48	32/25	47/57	25/23	1	77/43	35/35	9	68/48	33/33	None
8	55	F	Right	+	+	62/38	22/18	42/58	28/30	8	47/55	45/52	22	45/52	35/50	None
9	56	M	Left	+	+	.....	.....	.....	.....	..	62/68	55/45	29	62/62	52/47	None

* Indicates subsiding fistula symptom.
† Frequency 2048 not audible.

TABLE 2.—*Results of Corticotropin Treatment of Two Patients with Adhesive Otitis After Labyrinthine Fenestration Performed Seven and One-Half to Twelve Months Previously*

Patient	Age, Yr.	Sex	Ear with Fenestration	Fistula Symptom		Average Loss of Hearing, Db. at 512, 1024, and 2048 Cps										Subjective Improvement
				After Operation	During ACTH Therapy	Before Fenestration		Minimum After Operation		Mo. After Operation	Before ACTH Therapy		Mo. After Operation	After ACTH Therapy		
						Air	Bone	Air	Bone		Air	Bone		Air	Bone	
1	33	F	Right (9/2/49)	+	÷ *	52/67	15/12	50/60	17/17	3	58/58	43/35	12	55/52	23/28	None
2	48	F	Left (3/31/50)	+	÷	67/62	33/20	62/35	40/32	½	58/53	45/37	7½	58/53	37/30	None

* Indicates subsiding fistula symptom.

I. CORTICOTROPIN TREATMENT OF UNFENESTRATED EARS AND FENESTRATED EARS WITH LOSS OF POSTOPERATIVE IMPROVEMENT OF HEARING

The effects of corticotropin treatment appear in Tables 1 to 3, which give a survey of data on the patients. The acoustic function is illustrated by the average of the threshold values for 512, 1,024, and 2,048 cps, as introduced by Shambaugh [3] and since often employed. Main importance has been attached to hearing by air conduction, and only changes over 10 db. in this level are regarded as significant.

It appears from Table 1 that in all nine cases corticotropin treatment had no effect whatever, either on the ears not operated on or on those operated on. The improvement in hearing after fenestration, the time elapsed since the operation, the absolute hearing impairment, and the proportion of hearing by air conduction to that by bone conduction, as well as the patients' ages, proved of no importance in these cases. The hearing acuity, examined in all cases several months after treatment, remained unchanged. Patient 4 was later reoperated on with slight improvement only (cf. Table 5, Patient 1).

TABLE 3.—*Results of Corticotropin Treatment in a Patient with Otosclerosis* *

		Average Loss of Hearing in Db. at 512, 1024, and 2048 Cps											
		ACTH Treatment				Fistula Symptoms, Days After Operation		Days After Operation					
		Before		After				14		58		180	
Age, Yr.	Sex	Air	Bone	Air	Bone	14	180	Air	Bone	Air	Bone	Air	Bone
50	F	80/50	?/?	77/50	40/43	+	+ †	67/52	42/40	52/23	37/25	60/25	33/25

* Patient had rapidly progressive impairment of hearing in left ear for three months before treatment. Shortly after corticotropin therapy, labyrinthine fenestration was done in right ear, in which hearing had been poor for many years.
† Indicates subsiding fistula symptom.

In Table 2 the corticotropin treatment is seen to have had no effect whatever. Patient 2 (Table 2) was later reoperated on with a favorable result (cf. Table 5, Patient 2).

Another patient (Table 3) ostensibly had normal hearing in the left ear until about three months before admission, but since then there had been rapidly progressive impairment. The hearing in the right ear had been poor for many years. Table 3 shows that no improvement was observed in either ear immediately after the corticotropin treatment. Therefore, four days after the discontinuation of this treatment, fenestration was performed on the right ear, with immediate improvement of the hearing in this ear, while that in the left ear was still unchanged at this time. During the following six weeks or so, the hearing in both ears improved, but most markedly in the untreated left ear. On examination six months after the operation, the fistula symptom in the right ear had subsided, and the hearing in this ear was declining, while the improved hearing in the left ear remained unchanged.

Unfortunately, fenestration so soon after the corticotropin treatment has made this case somewhat difficult to assess, as some writers [4] claim to have observed improvement in the hearing of the ear not operated on in a number of cases after

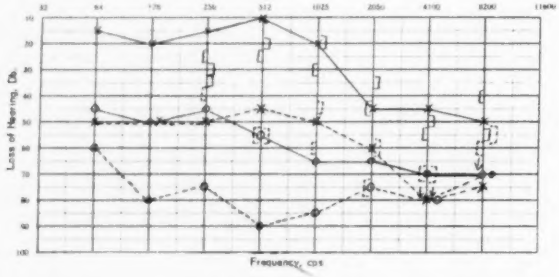
3. Shambaugh, G. E., Jr.: Acta oto-laryng., Supp. 79, 1949.
4. Holmgren, G.: Nord. med. **37**:151, 1949.

fenestration. Other writers,[5] however, declare that they have never seen unquestionable cases of this kind in investigations of much larger series. A review of the series from the Ear Department, Sundby Hospital,[6] bears out the view of the latter workers when Shambaugh's criterion is used as a basis.

It, therefore, seems to me most natural to explain the hearing improvement obtained in the ear without operation in this case as due to corticotropin, although improvement occurred strikingly late after discontinuation of the treatment. Audiograms taken before and after the treatment are shown in the Figure.

II. CORTICOTROPIN TREATMENT AFTER FENESTRATION

Improvement in hearing following fenestration may be lost again for various reasons, of which the most important are closure of the fenestra and development of fibrosis in the perilymphatic space, possibly with degeneration of the lateral semicircular canal. In addition, development of hydrops labyrinthi (Ménière's syndrome) is said occasionally to cause impairment of hearing, but only transitorily. Degeneration of the sound-perceiving cells may be disregarded in this connection,



Audiogram of patient with otosclerosis (Table 3). Broken lines indicate air-conduction loss, and brackets of broken lines indicate bone-conduction loss, before corticotropin treatment and labyrinthine fenestration. Lines and brackets of solid lines respectively indicate air- and bone-conduction loss 180 days after corticotropin treatment and labyrinthine fenestration. Lines with circles indicate values for right ear; lines with crosses, for the left ear.

because this generally develops far more slowly than the above processes, and perhaps in some measure may be considered as secondary to these.

Scandinavian writers [7] take closure of the fenestra to be the most frequent cause of postoperative loss of hearing, as, by the present technique, the fenestra has proved to close again in 10 to 15% within 6 to 12 months.[7c, d] Shambaugh, on the other hand, regards postoperative aseptic labyrinthitis as the greatest problem, and, with the use of his recently developed technique, he believes the incidence of closure of the fenestra is reduced to 1%.

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5. Shambaugh.[3] Lempert, J., cited by Jongkees, L. B. W.: Nederl. tijdschr. geneesk. **4**:4290, 1949.

6. Poulsen, I. P. V. (1950): To be published.

7. (*a*) Holmgren.[4] (*b*) Poulsen.[6] (*c*) Ewertsen, H.: Acoustic Function Before and After Operation, Arch. Otolaryng. **49**:393 (April) 1949. (*d*) Skoog, T.: Zur Frage der Indikationsstellung und Technik bei der chirurgischen Otosklerosebehandlung, Acta oto-laryng., Supp. 95, p. 24, 1951.

In my opinion the tendency toward closure of the fenestra is still the main problem, though prevention of aseptic fibrous labyrinthitis by no means is unimportant.

Ragan and associates[8] have shown experimentally that treatment with corticotropin has a greatly inhibiting effect on wound granulation.

Thus, corticotropin given immediately after fenestration should act on the processes leading to closure of the fenestra and possibly also inhibit or even prevent the development of fibrosis in the perilymphatic space to which injury or bleeding during the operation may give rise. To these may be added the influence on the vascular lesion present in the labyrinth in otosclerosis, which may perhaps reduce the risk of degenerative changes.

Postoperative corticotropin treatment was commenced eight days after the fenestration, and the drug was given in doses as previously indicated. Six patients have so far been treated in this way. Data on them appear in Tables 4 and 5.

It is seen from Table 4 that the hearing had improved considerably on the first examination; but it is impossible to judge the share of corticotropin treatment in the primary result.

The observation period is as yet too short for assessment of the late results.

The three patients mentioned in Table 5 had been treated with corticotropin without effect three to four months before the reoperation. It appears from the table that the first patient obtained no improvement in hearing after refenestration. No improvement was expected in this case because at the operation the fistula was found to be open but diminished (0.5 to 1.5 mm.), and the entire lateral semicircular canal showed fibrous degeneration.

The second and third patients, on the other hand, obtained even better hearing after refenestration than after the first operation, when the highest values are compared, even though bone-conduction hearing was best after the first operation. At refenestration the fistula of Patient 2 was seen to be completely closed by a thin white bone plate with a hard plug of fibrous tissue toward the lateral semicircular canal; but fibrosis extended no further into the perilymphatic space. In Patient 3 no trace was found of the labyrinthine capsule after the first fenestration. After these findings, it was not surprising that treatment with corticotropin before the refenestration had no effect.

III. FLUID RETENTION AND VARIATIONS IN UPPER AND LOWER HEARING LIMITS

All our patients gained weight during the treatment because of fluid retention. The maximum weight increase during the period of treatment varied from 0.7 to 6.2 kg. for the 12 patients mentioned in Tables 1 to 3; in five the gain was more than 4 kg. and in 7 it was under 3 kg.

To ascertain whether this might give rise to development or increase of labyrinthosis (hydrops labyrinthi), the upper and lower limits were tested by audiometry every other day during the treatment, and the patients were questioned daily about dizziness and tinnitus.

Examinations of the 24 ears revealed the following:

The upper limit for air conduction rose in 13 ears, remained unchanged in 6, and fell in 5. The variations ranged from +3,000 to —1,500 cps, but in 17 of the 24 examinations the variations were within ±500 cps.

8. Ragan, C.; Howes, E. L.; Plotz, C. M.; Meyer, K., and Blunt, J. W.: Proc. Soc. Exper. Biol. & Med. **72**:718, 1949.

TABLE 4.—*Results of Corticotropin Therapy Given from Seven to Fifteen Days After Labyrinthine Fenestration*

Patient	Diagnosis	Age, Yr.	Sex	Side of Operation	Fistula Symptom: 14 Days After Operation	Fistula Symptom: Last Examination	Average Loss of Hearing in Db. at 512, 1024, and 2048 Cps: Before Operation, Air	Before Operation, Bone	16 Days After Operation, Air	16 Days After Operation, Bone	Last Examination, Air	Last Examination, Bone	Observation Time, Mo.
1	Otosclerosis	37	M	Right	+	+	75/70	42/28	50/68	25/33	45/58	20/27	3
2	Otosclerosis	46	F	Left	+	+	57/52	40/33	52/32	37/30	52/28	33/27	1
3	Aplasia auris congenita	9	F	Left	+	+	68/68	33/40	.....	.....	68/42	32/22	1½

TABLE 5.—*Results of Corticotropin Therapy from Seven to Fifteen Days After Refenestration in Two Patients with Otosclerosis (Patients 1 and 3) and One Patient with Adhesive Otitis (Case 2)*

Patient	Age, Yr.	Sex	Ear with Fenestration	Fistula Symptom After Fenestration	Refenestration	Fistula Symptom After Refenestration	Average Loss of Hearing, Db. at 512, 1024, and 2048 Cps: Before 1st Operation, Air	Before 1st Operation, Bone	Minimum After 1st Operation, Air	Minimum After 1st Operation, Bone	Before Refenestration, Air	Before Refenestration, Bone	Minimum After Refenestration, Air	Minimum After Refenestration, Bone
1	43	M	Right (3/15/49)	+ *	Right (2/ 6/51)	÷	58/52	23/20	50/65	(25)/(25) †	60/60	38/43	55/60	42/47
2	48	F	Left (3/31/50)	+	Left (2/ 9/51)	÷	67/62	33/20	62/35	40/32	62/62	38/35	58/28	35/28
3	58	F	Right (1/17/50)	+	Right (4/19/51)	÷	80/68	23/22	57/58	33/32	70/62	38/(37)	42/65	37/48

* Indicates subsiding fistula symptom.
† Frequency 2048 not audible.

The upper limit for bone conduction rose in 11 ears, remained unchanged in 4, and fell in 8. In one ear the indication was uncertain. The variations ranged from +3,500 to —3,500 cps, being within ±500 cps in 14 of 23 ears examined.

The lower limit showed only insignificant deflections, ranging from +10 to —20 cps, being within ±5 cps in 18 of the 24 ears examined.

The two ears of some patients reacted differently, and there was no parallelism between the degree of fluid retention and the range of variation in upper or lower limit. None of the patients complained of dizziness. A few stated that they had slightly increased tinnitus, but the hearing was not influenced. Thus, there is no evidence to suggest that fluid retention should effect an increased intralabyrinthine pressure in patients with otosclerosis.

SUMMARY

A series of 15 patients, of whom 12 had otosclerosis, 2, adhesive otitis, and 1, aplasia auris congenita, was treated with corticotropin (ACTH) in doses corresponding to 20 to 30 mg. (Armour standard) intramuscularly four times daily for eight days. Twelve patients were treated without simultaneous fenestration. In six patients the treatment was commenced one week after fenestration.

The results achieved showed that this short course of treatment had no effect on ears with otosclerosis and adhesive otitis without operation when the impairment of hearing had persisted for several years. One otosclerotic patient, whose hearing had been impaired for only three months before the treatment, obtained considerable improvement, a fact that seems to suggest that early cases may respond favorably.

The hearing of fenestrated ears with closed fenestra or loss of postoperative hearing improvement for other reasons was in all cases uninfluenced by corticotropin treatment.

Treatment with corticotropin following fenestration gave good primary results; but the observation period is still too short for assessment of the late results.

The treatment had no troubling side-effects; more particularly, it caused no undesired acoustic or vestibular reaction in spite of considerable fluid retention in most of the patients.

ANESTHESIA FOR LONG OR TECHNICALLY DIFFICULT OPHTHALMOLOGIC AND OTOLARYNGOLOGIC SURGICAL PROCEDURES

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AND
JOHN F. TOLAN, M.D.
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IN OPHTHALMOLOGIC and otolaryngologic surgery one encounters some of the most difficult procedures in anesthesia. This is particularly true if the requirements of the patient, the surgeon, and the anesthesiologist are to be satisfied. Regional analgesia may be employed satisfactorily for short superficial procedures, since the patient will usually tolerate an uncomfortable position as well as manipulation discomforts for limited periods. However, during long and/or tedious procedures requiring deep manipulations of the face, nasal pharynx, oral pharynx, pharynx, larynx, or trachea, the constant cooperation of the patient must not be relied on. Restlessness and motion at a crucial point by a patient who was thought to be cooperative may prove disastrous to the patient or to the operative result being sought. Likewise, the administration of an intravenous, rectal, or inhalation anesthetic to a patient in whom regional analgesia was started and whose airway has been made inaccessible by the drapes or the surgery may be equally catastrophic.

Many physicians practicing ophthalmology and otolaryngology at the present advocate performing long and/or tedious surgical procedures with the patients under basal narcosis plus local block. If the patient becomes restless, more sedation is added by the rectal or intravenous route. Should these measures fail to quiet the patient, general anesthesia, usually in the form of intravenously administered thiopental (pentothal®) sodium, is instituted. Various gadgets are used to hold the patient's head. In most of these circumstances, little if any attention is paid to the patient's airway or to the hypoxia which must result from heavy sedation, the anesthetic agent, or the combination of the two. An oral airway at times is inserted blindly under the drapes. To add to the difficulty, many of the procedures are performed in a dark or poorly lighted room so that the surgeon's head mirror or lamp may be more effective and, therefore, the patient's color and respiratory exchange cannot be adequately visualized.

Because of the above-mentioned circumstances and the difficulties experienced by the surgeon and the anesthesiologist, we have agreed that in all long procedures or technically difficult short procedures anesthesia with an endotracheal tube to insure the patient an unobstructed airway and adequate oxygenation through the

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entire surgical period shall be established before the patient is aseptically prepared and draped and the surgery is started.

The choice of the agent in these cases is immaterial provided the airway is established by intubation and the necessary precautions concerning explosions are observed. Most ophthalmologists and otolaryngologists prefer to work with a head mirror plus a lamp or a head lamp. These items are seldom explosion proof. Also, not uncommonly a dental drill or an electric cautery is necessary for the operative procedure. Pounding of chisels or the clicking of instruments is another source of sparks. The aforementioned problems make a nonexplosive agent or combination of nonexplosive agents the anesthetic of choice. Also, in the present era, when thiopental sodium is the patient's preference the physicians in private practice are met with the problem of satisfying this desire. Therefore, the technique presented here for performing otolaryngologic and ophthalmologic surgical pro-

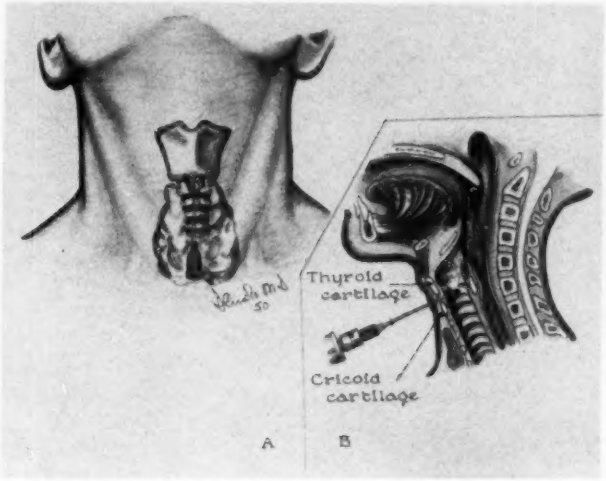

Fig. 1.—Anatomy of the trachea with needle in place for transtracheal injection.

cedures necessitating general anesthesia was devised to meet the requirements enumerated. While we do not claim it to be the only method, it is highly satisfactory, and other physicians may find some of the steps in its execution helpful.

It must be pointed out that cataract surgery is performed with the patient under local anesthesia in order to prevent straining or coughing during the anesthetic or postanesthetic period. In such surgery, even if an endotracheal tube was not placed and general anesthesia employed, there may be some straining, coughing, or vomiting during the anesthesia or the immediate postoperative course in a certain percentage of cases, which may be detrimental to the desired result.

TECHNIQUE

Preoperative sedation is ordered only after the patient is visited by the anesthesiologist. The drugs used for premedication depend on the individual preference of the anesthesiologist and the surgeon. Overpremedication should be avoided. The preanesthetic visit is essential, as the

anesthesiologist may gain the confidence of the patient, and this is the first step in any successful anesthetic procedure.

When the patient arrives for surgery, he is again reassured by the anesthesiologist that he will have no trouble with the anesthetic. He is placed on the operating-room table, and 2 to 3 cc. of 2% tetracaine (pontocaine®) hydrochloride solution is injected transtracheally (Fig. 1). This technique is a rapid method of locally anesthetizing the upper trachea, larynx, and posterior pharynx and consists of the following steps.

1. The technique is explained to the patient, and he is instructed not to cough, swallow, or talk until told to do so.

2. The head is hyperextended (Fig. 2).

3. The cricoid-thyroid membrane is identified. It is the membrane between the cricoid cartilage and the thyroid cartilage. The cricoid cartilage is easily identified since it is the only complete cartilage of the trachea and as such is firm and has the feel of a wedding ring (Fig. 1).

4. If the anesthesiologist is right-handed, the index finger of the left hand marks the spot of injection (Fig. 2).

Fig. 2.—Transtracheal injection. Note the position of the anesthesiologist and his left hand palpating the position for insertion of the needle.

5. The skin is aseptically prepared at the site of entrance of the needle. A 1½-in. (3.8 cm.), 20 or 21-gauge Huber point Lok-needle attached to a 5 cc. Lok-syringe containing 2 to 3 cc. of a 2% tetracaine solution is inserted in the midline, with the bevel of the needle pointing cephalad, and is advanced perpendicularly to the skin until it is felt to "pop through" the cricoid-thyroid membrane. There is a feeling of less resistance at this point.

6. Aspiration of air is attempted to confirm the fact that the bevel of the needle rests within the lumen of the trachea.

7. Again the patient is cautioned not to cough, swallow, or talk.

8. The contents of the syringe are quickly emptied and the needle withdrawn.

9. The patient will automatically cough and feel that he cannot get his breath. He should be reassured and urged to cough violently so as to spread the anesthetic solution.

Now the patient's nostrils, turbinates, mouth, and upper pharynx are sprayed with 2% tetracaine hydrochloride with an ordinary atomizer (Fig. 3). The blood-pressure cuff is applied, the patient's position on the table secured with a knee strap and wrist cuffs, and an intravenous drip with at least an 18-gauge needle started. These maneuvers require 5 to 10 minutes, depending on the speed of the anesthesiologist, and allow the 2% tetracaine solution instilled transtracheally to establish adequate anesthesia of the nose, mouth, pharynx, larynx, and trachea.

A six-tenths (0.6) per cent solution of thiopental (pentothal®) sodium is administered slowly by the drip method until the patient's lid reflex and corneal reflex are absent. If the operation is not in the region of the nose or nasopharynx (i. e., fenestration, eye surgery), a No. 32 or 29 French endotracheal tube lubricated with 1% dibucaine (nupercaine®) hydrochloride ointment (ordinary nupercainal®) is passed through the nose (Fig. 4). Two or three attempts at blind intubation are made, and if they are unsuccessful, the laryngoscope is used to pass the tube through the larynx. Blind intubation is successful in approximately 80% of these cases. It is seldom

Fig. 3.—Spraying of the nostrils and turbinates.

Fig. 4.—Method of blind intubation. Note that the chin is held up with the left hand as the endotracheal tube is slowly advanced through the floor of the nasopharynx with the right hand. The anesthesiologist listens to the breath sounds as the tube is advanced so that he may ascertain when he is approaching the larynx.

necessary to depress the patient's respiration with large intravenous doses of thiopental to pass the endotracheal tube. If the patient is not relaxed adequately for use of the laryngoscope, 2 to 4 cc. of d-tubocurarine is administered intravenously. Once the tube is in place, the pharynx and the opening of the esophagus are packed with vaginal gauze packing to assure a closed system and to prevent blood or secretions from entering the tracheobronchial tree. If the operation involves the nose, nasopharynx, or sinuses, an endotracheal tube with an inflatable cuff is inserted through the mouth, the method of anesthesia described above being used. The cuff is inflated to

prevent blood and secretions entering the trachea and lungs. The endotracheal tube is then connected to the anesthetic machine, and the patient is allowed to breathe a mixture of approximately 60% nitrous oxide and 40% oxygen (Fig. 5). The 0.6% thiopental sodium drip solution is replaced with a solution of 5% dextrose in distilled water. In many cases the nitrous oxide-oxygen mixture is adequate for the procedure from this point. Should it be inadequate, supplementary anesthesia is given with the following drugs individually or in combination: (1) 0.2% thiopental sodium intravenously; (2) d-tubocurarine, 0.5 (10 units) to 1 cc. (20 units), intravenously and repeated in 10 minutes if inadequate; (3) morphine sulfate, ⅛ grain (8 mg.) intravenously, repeated if necessary in 15 minutes.

Fig. 5.—Patient's head resting on "donut."

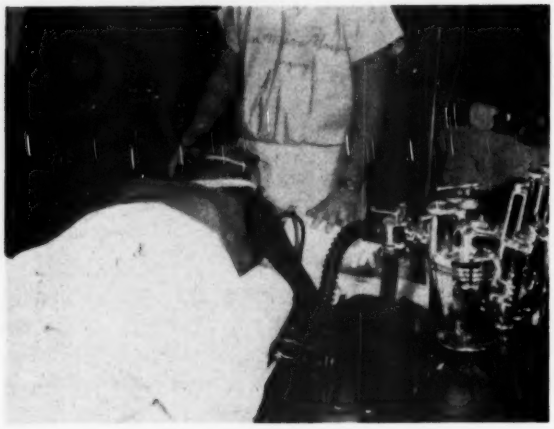

Fig. 6.—Anesthetic machine attached to endotracheal tube and fluids running intravenously. Note that the anesthetic machine and the anesthesiologist can be well away from the surgical field so that the surgeon has freedom of motion.

The patient's head is placed on a "donut" (Figs. 5 and 6). This gives stability to the head and allows the surgeon actually to position it. The surgical procedure is then carried out. The anesthetic machine is placed at a distance from the surgical field so that the surgeon and his assistants are not hindered (Fig. 6).

At the end of the surgical procedure, the patient is flushed with oxygen. Tracheal aspiration through the endotracheal tube is performed, pharyngeal aspiration carried out, the pharyngeal pack removed or the cuff deflated, and the endotracheal tube removed. At this point most patients are responding. If, however, the patient is still anesthetized, it is not uncommon when a nasotracheal tube has been employed to remove the pack and leave the tube in place until the patient responds. On the other hand, if the patient has responded and there is a question of maintaining an airway, such as may be the case after incision and drainage of neck abscesses when there is edema of the structures of the pharynx, the tube often is allowed to remain in place until the edema subsides. If it is desirable to remove the tube, as in the case of most oral intubations, and the patient has not responded, then an oral airway should be inserted after the tube's removal to assure an adequate airway during the immediate postoperative period. We routinely give what we call the dramamine® (dimenhydrinate) routine. This consists of 50 mg. of dramamine® intramuscularly on call to surgery, 50 mg. given intramuscularly immediately on return from surgery, and then 50 mg. given intramuscularly every four hours postoperatively for four doses. This routine has greatly decreased postoperative vomiting and dizziness. In selected cases the dosage of dramamine® may be continued for a longer period if indicated.

Surgical Procedures Performed in 114 Cases

Eye Surgery	
Muscle resection	14
Suturing of eyelid	1
Removal of lacrimal sac	2
Enucleation	5
Removal of foreign body	6
Removal of cyst of eye	2
Plastic on eyelids	2
Retinal detachment	6
Iridectomy	2
Ear	
Fenestrations	33
Mastoidectomies	5
Neck	
Radical neck dissection	3
Tonsillectomy and adenoidectomy (adults)	23
Facial Plastics	
Nose	4
Jaw resections	8
Total	116

RESULTS

Fifteen different surgical procedures were performed in 116 cases (Table). Seventy-two patients were intubated by the nasotracheal technique and the remainder by the orotracheal method. All the patients received the transtracheal injection of 2% tetracaine hydrochloride. Six patients in this series were returned to their rooms with a nasotracheal tube in place. There were nine cases of postoperative hoarseness attributable to the anesthetic procedure.

COMMENT

As has been pointed out previously, we feel that the most satisfactory method of anesthetizing a patient for a long and/or technically difficult surgical operation of the head or neck requires the establishment of a clear, easily maintainable airway prior to the beginning of the surgical procedure. The insertion of an endotracheal tube is the answer to this problem. Once this maneuver has been accomplished, the anesthesiologist has complete control of the airway and may supplement any inadequacy in the respiratory exchange. The anesthesiologist must not rely on

the patient who is heavily narcotized with opiates and barbiturates and who has only a small degree of consciousness to oxygenate himself adequately. Even if he should not partially or totally obstruct his airway, a chronic type of oxygen want may occur from depression of the rate and/or depth of the respirations. Some argue that even though the respiratory rate and/or depth of the respirations are decreased, the patient does not have anoxia because the metabolic rate has been decreased and therefore the oxygen demand is not as great. This argument is to be questioned. Perhaps one of the causes for excessive bleeding when there is no obvious reason is the result of an underlying chronic oxygen want. We have observed at various institutions that surgeons who perform fenestrations with the patient under heavy narcosis plus local anesthesia often make the following comment to the visitors: "This patient is oozing more than usual." In our 33 fenestrations we have found that even though we use a minimal amount of local anesthetic solutions with vasoconstrictors the amount of bleeding which occurs has never been excessive nor has it complicated either the surgical procedure or the results. Also, we and others feel that whenever respiratory rate and/or depth of the respirations are markedly decreased without an increase in the oxygen intake in the respired atmosphere a chronic type of oxygen want occurs.

The route of intubation, i. e., nasotracheal versus orotracheal, and the type of endotracheal tube, i. e., plastic or rubber, with or without an inflatable cuff to be used, should be the decision of the person placing the tube. The choice of the route depends on the following [1]:

1. The area in which the surgeon is operating.

2. The skill of the anesthesiologist.

3. Nasal deformities. If there are apparent nasal deformities or it is difficult to pass the nasal tube, the oral route should be employed.

4. Length of time the endotracheal tube is to be left in place. If an endotracheal tube is to remain in place to maintain an airway after a surgical procedure, the nasal route is the more comfortable for the patient and is easier to care for from the nursing standpoint; for example, in incision and drainage of an infected neck, the endotracheal tube is often used postoperatively for 12 to 24 hours to maintain the patient's airway until the edema has subsided.

5. The depth of anesthesia. Nasotracheal intubation may be performed in lighter planes of anesthesia than oral intubation.

6. Preexisting nasal infections. When these are present, it is best to avoid nasotracheal intubation. However, it is usually agreed that the route of intubation in the normal patient has little influence on the incidence of postoperative respiratory complications.

The type of tube to be used depends on the following [1]:

1. Route of intubation. In general, if a cuffed tube is to be used, the oral route should be chosen because of increased chance of trauma to the nasal mucosa. This choice is questioned by some.[2]

2. Size of the endotracheal tube. If it is advisable to have a large endotracheal tube in place to facilitate tracheal suction, the oral route is to be preferred since usually the nasal cavity will accommodate only the smaller-lumened endotracheal tube.

3. Correct placement of the tube. It may be difficult to place the cuffed tube correctly in the trachea so that the cuff lies inside the cords, unless the cords are visualized. This difficulty immediately removes many of the advantages of nasal intubation.

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1. Gillespie, N. A.: Endotracheal Anesthesia, The University of Wisconsin Press, 1948.

2. Tovell, R. M.: Proc. Staff Meet., Mayo Clinic, 1936. Grimm, J. E., and Knight, R. T.: Anesthesiology, 1943.

4. Route of intubation. The rubber endotracheal tube may be preferable to the plastic endotracheal tube when the nasotracheal route is to be employed, since it is stiffer and thus maintains its curve; however, if the plastic tube is placed in cold water or ice prior to its use, it becomes stiff and maintains its curve well. The curve of the tube should approximate that of the nasopharynx and pharynx if the nasal approach is to be successful.

The choice of drugs for premedication depends on the individual preference of the anesthesiologist or the surgeon. We prefer to premedicate our patients as follows:

1. One and a half to 3 grains (0.1 to 0.2 gm.) of a barbiturate is administered the night before surgery to assure the patient a good night's sleep. We usually omit barbiturates the morning of surgery, but, if desirable for the nervous patient, 1½ to 3 grains (0.1 to 0.2 gm.) of pentobarbital (nembutal®) sodium may be administered. The rectal or intravenous route is preferred in such cases to the oral for three reasons. First, absorption is assured. It is a known fact that if barbiturates administered orally are not given at least one-half to one hour before the opiates, they may not be emptied from the stomach, since opiates cause pylorospasms. Also, even without the influence of the opiate, a tense patient who is apprehensive may not empty his stomach for a variable time preoperatively. Second, when oral medications are given by a nurse, the amount of water involved may vary from a half ounce to a glass depending on such factors as the patient's ability to swallow, etc. Third, barbiturates given orally are best absorbed from the alkaline medium of the small intestine; if they are retained in the stomach, complete absorption and the height of their effect may not be realized until the middle of the surgical procedure or after it. This in turn may cause serious respiratory depression when the anesthetic agent is added to the effect of the barbiturate.

2. One-sixth to ¼ grain (10 to 15 mg.) of morphine sulfate or a comparable substitute with 1/100 grain (0.6 mg.) of atropine is given intramuscularly three-quarters of an hour before surgery. Atropine is preferred to scopolamine because of its stronger blocking action on the vagus nerve. This is important because the thiobarbiturates, in which classification thiopental sodium falls, are notable for making vagal reflexes, particularly the laryngeal reflex, hypersensitive.

3. Fifty milligrams of dimenhydrinate (dramamine®) is administered intramuscularly when the patient is called to surgery in order to establish its effect by the time surgery has been completed.

The method of transtracheal injection is not a new procedure. It was described by George Conuyt and published 30 years ago by Labat in his book,[3] and a recent article by Bonica has appeared on this subject.[4] It must be stressed that more than 3 cc. of the local anesthetic solution should not be injected transtracheally if reactions are to be avoided. We prefer 2% tetracaine, but a 5% cocaine solution is equally effective. In approximately 4% of the cases local anesthesia of the larynx and trachea is unsatisfactory and the cords must be sprayed directly with the local anesthetic solution.

The essence of this paper is to stress the placement of the endotracheal tube and not the choice of the anesthetic agent, the type of tube, or the route of placement of the tube. However, it should be stressed that trauma to the larynx or pharynx during the insertion of the endotracheal tube is to be avoided if at all possible. Granuloma of the larynx occurs with enough frequency after intubations so that many feel that trauma from the endotracheal procedure as well as from the tube itself may be one of the predisposing factors which lead to granuloma formation. We

3. Labat, G.: Regional Anesthesia, London and Philadelphia, W. B. Saunders Co., 1930.

4. Bonica, J. J.: Transtracheal Anesthesia for Endotracheal Intubation, Anesthesiology 6:736-738 (Nov.) 1949.

have chosen thiopental (pentothal®) in this series of cases because it is a pleasant anesthetic from the patient's standpoint and is nonexplosive. We do not hesitate to change the anesthetic agent if we feel that the physical status of the patient warrants it. However, we do not use cyclopropane when the surgeon is going to inject or topically apply epinephrine per se, or local anesthetic solution containing epinephrine since the combination of epinephrine plus cyclopropane predisposes to ventricular fibrillation. It is not uncommon for us to employ d-tubocurarine previous to the intubation for relaxation in order to conserve the amount of thiopental necessary for the intubation and the surgical procedure. Also, Claude Bernard demonstrated experimentally in 1856, and recently Burstein, Jackson, Bishop, and Rovenstein demonstrated clinically, that curare depresses certain autonomic reflexes.[5] The vagal reflex is one of these. Therefore, it is reasonable perhaps to advocate the use of curare in 2-cc. to 4-cc. doses previous to intubation under light thiopental anesthesia to block the vagovagal reflexes. Often during the anesthesia, if we find we are going to exceed 1.5 gm. of thiopental, we give morphine intravenously, ⅛ to ¼ grain (8 to 15 mg.) in divided doses, to decrease the amount of thiopental required. Other opiates may be used intravenously instead of morphine to accomplish the same end-result, i. e., the requirement of less thiopental, but it should be emphasized that if synthetic derivations are employed, particularly meperidine (demerol®) hydrochloride, much smaller doses than those advocated for morphine should be injected if respiratory arrest is to be avoided. For example, if 75 mg. of meperidine is assumed to be equivalent to ⅛ grain (8 mg.) of morphine sulfate, the initial dose of demerol® to be given intravenously with the patient under thiopental anesthesia should be reduced to 20 or 40 mg. if respiratory arrest is to be avoided.

Nupercaine® ointment (nupercainal®) is spread thinly on the endotracheal tubes as a lubricant as well as a topical anesthetic to anesthetize the structures of the nose, mouth, pharynx, larynx, or trachea with which it comes in contact. This is a great help with light anesthesia if the cough reflex is to be controlled or abolished during slight manipulation of the head or movement of the tube. Some object to nupercaine® ointment because they feel it is irritating to the mucosa and causes increased secretions and that oily substances should not be introduced into the tracheobronchial tree if at all avoidable since they may cause oil pneumonitis. Should the latter argument be a valid one, nupercaine® may be incorporated in an aqueous base. The formula [6] for such a mixture is (1) nupercaine,® 24 gm.; (2) tragacanth, 50 gm.; (3) glycerin, 400 cc.; (4) zephiran® (benzalkonium chloride) concentrated, 1 cc., and (5) water, 1,200 cc. This formula makes a fluidy 2% nupercaine® ointment.

All patients in whom this type of anesthesia was administered tolerated the anesthetic procedure well. There were no postoperative complications attributable to anesthesia other than hoarseness in an insignificant number of patients. Postoperative rounds were made within the first 24 hours and then every other day until the patients left the hospital. Every patient was asked if he liked the anesthetic. All responded favorably, but 22 patients raised a slight objection to the stick in the neck from transtracheal injection. We therefore tried starting the administration of thiopental previous to this procedure and found that it was satisfactory providing

5. Burstein, C. L.; Jackson, A.; Bishop, H. F., and Rovenstein, E. A.: Curare in the Management of Autonomic Reflexes, Anesthesiology 4:409-421 (July) 1950.

6. Leigh, M. B.: Private communications to the authors.

the patient was not anesthetized beyond the analgesic state, i. e., that the lid reflex had not been lost, but merely slowed, with the thiopental. In this stage of analgesia the patient has not lost consciousness, will follow instructions, will cough vigorously and will have no recollection of the tracheal injection after recovery from the anesthesia. Two to three minutes must elapse after the transtracheal injection before intubation is attempted; otherwise, the local anesthetic agent will not have time to anesthetize the trachea. If the transtracheal injection is delayed until thiopental analgesia is established, careful timing of the administration of the thiopental and the stage of anesthesia is a necessity.

SUMMARY AND CONCLUSIONS

Irrespective of the anesthetic agent or technique employed, an endotracheal tube should be placed in all prolonged and/or technically difficult operations of the head and neck before the patient is aseptically prepared and draped and the operation started.

The methods advocated above have satisfied the patient, the surgeon, and the anesthesiologist and have avoided the use of explosive anesthetic agents in operative procedures which often entail the use of electrical mechanical devices, pounding of chisels, and the clicking of instruments in the same region as that which must be used for the administration of the anesthetic.

ABSTRACT OF DISCUSSION

DR. PAUL B. MACCREADY, New Haven, Conn.: Dr. Moore has described an excellent method for the giving of thiopental intratracheally. He has laid great stress on having a free airway, which, of course, is fundamental. There is one modification in his technic which is different from that normally used or extensively used and which I would question, namely, the injection of the tetracaine (pontocaine®) through the cricothyroid membrane. I have talked with different anesthetists, some of whom have used it and some who know about it, and they all say it is a very efficient procedure. I recall a death from tetracaine that has been published in the *Laryngoscope* for June, 1946. The patient was being anesthetized for bronchoscopy, and I do not think that there was sensitivity to tetracaine because the patient had been anesthetized two weeks previously and a bronchoscopy performed. After anesthetizing the pharynx and the larynx, the resident injected 3 cc. of tetracaine into the trachea through a catheter. The patient immediately went into shock and convulsions and was dead in 20 minutes. I should like to suggest that there was a sudden flash absorption of the tetracaine, which, of course, is toxic, although not as much as cocaine.

I think that if the procedure which Dr. Moore describes is used universally sooner or later we will have more incidents of that nature. The anesthetists are all in favor of this intratracheal type of anesthesia, and many of the surgeons do not feel the same way about it. Dr. Moore has reported 33 fenestrations. I have a list of 150 fenestrations done a little differently. When we originally started, we were dependent on nurse anesthetists, and a nurse anesthetist helped me work out this plan. We give the thiopental as an infusion and then insert a metal airway; a catheter is inserted through the nose into the hypopharynx and oxygen is given through that. Nitrous oxide is given through a mask held on the face. I have a notion that when I start working the anesthetists cheat a bit and give the nitrous oxide through the catheter rather than hold the mask on the face. They do not like this type of anesthesia. It is harder for them, but it is an operation which does not involve the airway, and I should like to point out I have done some operations under intratracheal anesthesia and it is my distinct impression that there is less bleeding with the former method.

I noticed a report of tonsillectomies performed with the use of the anesthesia under discussion. I believe that the Syracuse Clinic reported doing tonsillectomies using intratracheal thiopental-nitrous oxide anesthesia about a year ago, and I see that there is another report on the program.

As a universal procedure, it is questionable. I do not use it. I use a Sewell-Davis mouth gag, with the patient in the Rose position; aspiration is not a factor, and we have as free an airway as it is possible to get and frequently look right down into the larynx.

When the thiopental is given intratracheally, the big difficulty is that occasionally severe laryngospasm occurs after the patient has been sent back to the ward. If the hospital has a recovery ward, where these patients can be watched carefully immediately after operation, that is one thing; but I think that in the average hospital there will be difficulty sooner or later with the procedure.

Dr. Moore gave a detailed description, and I think that in his hands this would be a most excellent method.

Dr. P. L. Munday, Camp Carson, Colo.: In Camp Carson, in Colorado, we are using a good part of this procedure. What prompted me to come up here was the use of the tetracaine. I have seen a death occur from the use of tetracaine, and it was not very pleasant. I was anesthetizing a young girl who came in for an elective procedure. After I instilled the tetracaine to have her cough and gargle it, the next thing I knew she was not talking to me any more. She started into clonic and then, finally, tonic contractions. In spite of what we could do, we lost the patient. We did not have any barbiturates on hand for intravenous administration; we should have had. We tried to use ether to help her get over the attack, but it was of no avail.

My understanding is that tetracaine is about four times as toxic as cocaine and not less toxic, unless it is used with epinephrine to cut down its absorption. Tetracaine would be very safe if we could use a few drops of epinephrine with it or a few drops in the trachea first, followed by pontocaine or by cocaine itself instead.

Dr. Joseph J. Kemler, Baltimore: It was my impression that the Veterans Administration have cut out tetracaine altogether, at least the branch in New York, where I operated. I was told that they cannot use tetracaine of any sort at any time. I have been using it since it became available. I find that the only trouble with it is that it must not be used in allergic patients or in asthmatic conditions. I had a patient who had asthma on whom I was going to do a submucous resection. I told the nurse preparing the patient not to use tetracaine. When I came to operate, the patient was already prepared and the nose had been sprayed with tetracaine. He had a severe asthmatic attack. Of course, I did not operate that day. Fortunately, the man recovered, and I operated the next day.

In my office I never use tetracaine unless I know that the patient is not allergic and has had no asthma at any time. In some places they do not use thiopental sodium unless the trachea is intubated. When I was operating at Israel for tonsillitis and other conditions, they said, "You cannot use thiopental (pentothal®) sodium unless we intubate first."

I want to ask the essayist whether, instead of injecting tetracaine into the trachea, it could be used in aerosol form with oxygen through the mouth, just for the last 10 or 15 minutes, in the same amount, or even a less amount, until the lips get numb.

Dr. Frank D. Lathrop, Boston: There is frequently a difference of opinion in any discussion concerning a method of anesthetization whether it be for otolaryngologic or for general surgery. Recently, I have heard much about tetracaine and little about cocaine. I still use cocaine when I think it is indicated even though I have had the misfortune to have had a death from the use of cocaine. However, I have used tetracaine in the same manner as that described by Dr. Moore since 1942.

This method has been used for all bronchoscopies performed under local anesthesia and, on occasion, in preparation for the insertion of an endotracheal tube. I do not believe that more reactions occur with tetracaine when properly used than with any other local anesthetic agent. If a reaction does occur, perhaps it will be severer than with other agents, but preparations should always be made to counteract such reactions, if necessary, regardless of the agent used.

We use endotracheal anesthesia for the majority of otolaryngologic operations at the Lahey Clinic. I cannot recall a case of major surgery in recent years in which it has not been used. We like endotracheal anesthesia because the depth of anesthesia can be controlled satisfactorily. In otolaryngology the anesthesia need not be deep; preferably it should be light when a general anesthetic is administered for tonsillectomy. I prefer to have the cough reflex present in such circumstances and will stop operating if this reflex is lost and continue when it recurs as the depth of anesthesia is lightened.

During the past two years we have used endotracheal ether for tonsillectomies when general anesthesia is desired. We have not had any difficulty even though the tonsillectomies were performed with the patient in the sitting position. We have not experienced difficulty with aspiration of secretions in this position either before or since an endotracheal tube has been employed.

DR. A. D. RUDO, Baltimore: I wanted to answer Dr. Kemler's statement with a description of a method of nebulizing anesthesia that we were using at the Veterans Hospital in the Bronx. It consisted of a DeVilbiss No. 44 nebulizer, to which is attached about 2 in. (5 cm.) of rubber tubing and which is in the mouth. This tubing would enable us to move the nebulizer farther back as anesthesia proceeded so that by the time it was almost completed the rubber tube would be sitting on the arytenoid cartilages. We used 5% cocaine, but we did not add any epinephrine to it.

The reason that the use of dibucaine was stopped by the Veterans Administration in New York was because Dr. Rovenstine had had several deaths from this drug at Bellevue and he felt that its use was not indicated, as well as the solution which we had been using prior to that (Forestie's).

This anesthesia is extremely practical because the patient can anesthetize himself while the operator is working. Of course, the operator has to supervise occasionally, to see that the patient in his premedicated state will advance the rubber tubing and the nebulizer farther down. A full description of this method was given by one of our men, who devised it and reported it in *Annals of Otolaryngology* in December, 1949.

DR. JAMES COSTEN, St. Louis: There is one point that Dr. Moore made which I think might be the solution to why tetracaine works safely for some patients in a universal way and for others very differently. He said that some time before the morphine and atropine were given a barbiturate was administered by mouth. I think that is the most important protection that he could give his patient against a reaction to tetracaine.

In our clinic, at the McMillan Hospital, a barbiturate is given routinely before all the nose and throat procedures. As the patients come into the endoscopy room, the assistant starts spraying the larynx with tetracaine, whether the patient will be given thiopental or not. I have never seen any reaction to the tetracaine there. I cannot say too much about the intratracheal aspect, because I am particularly fond of local anesthesia. I use local anesthesia for all fissures, with the same premedication that Dr. Moore mentioned, namely, barbiturate, and morphine and atropine afterward. For laryngectomy I use the same, unless the patient begins to get restless toward the end of two hours, in which case I give thiopental. I have never had any trouble with the use of the latter. I think the protective action of premedication with barbiturates is the most important point in the whole discussion.

DR. HENRY M. SCHEER, New York: I think it was just about two years ago in this very room when we had a discussion on this subject of tetracaine versus cocaine anesthesia and its use in bronchoscopy. It was in a discussion of Dr. Dan Cunning's paper on carcinoma of the larynx. He was very much in favor of the use of tetracaine. The discussors brought up this controversial question of tetracaine versus cocaine, and the preponderance of opinion, if I recall correctly, was that cocaine was somewhat safer than tetracaine. We know that when a laryngectomy is being performed, two or three minims of cocaine is dropped into the trachea before it is opened. This measure ensures good anesthesia and prevents cough when the trachea is opened. It is possible that the use of a drop or two of cocaine may prevent those accidents occasioned with use of tetracaine.

DR. JOSEPH J. KEMLER, Baltimore: I have had no experience with aerosol 2% pontocaine,® but the late Dr. Dale Davis, of Washington, used it, and I think he had very good results. When I operate with the use of tetracaine in my office and in the hospital, I always give pentobarbital sodium (nembutal®), 1½ grains (0.1 gm.), one hour before the tetracaine is inserted.

DR. DANIEL C. MOORE, Seattle, Wash.: We prefer the transtracheal method of anesthetizing the trachea, larynx, and posterior pharynx for two reasons. First, it is less time consuming and technically less tedious. Second, the exact amount of the local anesthetic solution is accurately

measured. The latter reason is especially important since often the task of anesthetizing the respiratory tract is the duty of the intern or resident. Often, in an attempt to establish perfect anesthesia, he is too generous with the local anesthetic solution if the gargle, swab injection method using the laryngeal mirror is attempted.

The question concerning the fact that more deaths occur from placing tetracaine in the trachea than from cocaine presents an interesting problem. Milligram for milligram, tetracaine is more toxic than cocaine. However, since the solution of tetracaine used is one-half to one-fifth as as strong as the cocaine solution, it is actually less toxic. Reactions to 2% pontocaine® and 5 or 10% cocaine do occur, irrespective of the technique used, and I have seen them occur. Nevertheless, to date we have never had a patient who died from a severe toxic reaction. It cannot be too strongly emphasized that when a reaction occurs the first thing to be done for the patient is to establish a clear airway and give oxygen by bag and mask, using this setup as a means of artificial respiration if necessary. Barbiturates should be given to control convulsions but only after oxygen therapy has been instituted. When a reaction occurs, the anoxia is the most important factor to combat since it is probably one of the important inciting causes of cardiac arrest. It is not uncommon for a physician to attempt to mix and administer a barbiturate to the patient when a toxic reaction occurs before instituting any other therapy. In doing so many valuable minutes are lost while the patient is suffering severe anoxia. Therefore, I cannot too strongly emphasize the importance of oxygenation of the patient as the first step in treating a toxic reaction.

The use of the endotracheal tube in procedures on the head and neck has always been a controversial issue. Nevertheless, from the average anesthesiologist's viewpoint it is more satisfactory to establish an adequate airway before the draping and positioning of the patient than after the surgery has begun.

Anoxia and hypoxia are known to cause a constant oozing from the tissues, and intubation is good insurance against anoxia or hypoxia states. Oozing often makes an operation in a well-defined surgical field most troublesome, and this in particular applies in the fenestration operation.

I feel that tonsillectomy in adults should be performed under local anesthesia, but if the surgeon desires general anesthesia, then the placement of an endotracheal tube is imperative.

ROLE OF ALLERGY IN OTOLOGY

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CLINICAL observations indicate that many body changes classified as allergic cannot be explained purely on the basis of antigen-antibody reactions. Autonomic dysfunctions, including psychic changes, vasomotor reactions, and endocrine imbalances, are equally important in the production of this altered reaction known as allergy. The phylogenetic concept of allergy as presented by Williams [1] offers a refreshing approach to the scope of allergy. The statement by Forman [2] that allergy is the sum total of unfavorable stresses and strains which the environment places upon the individual indicates a broadening of the concept. The appreciation of allergy in the various fields of medicine has altered the diagnosis and treatment in many diseases. This change has been very apparent in otorhinolaryngology.

It is the purpose of this presentation to evaluate the role of allergy in otology. The material presented was obtained from a partnership practice limited to otology. The partnership consists of Dr. Kenneth Day and myself. The material covers otological conditions caused or complicated by allergy.

In order to arrive at a percentage figure which would be representative of the allergic part of our practice, new cases observed during 1950 were used. During this period 727 cases had sufficient observation to be of statistical value. The order of presentation will be that of conditions involving the external, middle, and inner ear.

CONDITIONS INVOLVING THE EXTERNAL EAR

During 1950, 47 cases of chronic otitis externa were observed. The causation in 39 cases was thought to be traceable to bacteria, fungi, or both. The condition in eight cases was diagnosed as allergic manifestation. Some of the allergic patients responded to treatment with antihistaminic creams or ointments; others required allergic investigation.

CONDITIONS INVOLVING THE MIDDLE EAR

One of the commonest middle ear conditions seen in our practice is chronic secretory otitis media, also known as otitis media with effusion. This diagnosis was made in 111 cases during 1950; in 87% of these cases the condition was classified as allergic in origin. This percentage compares favorably with the results of a four-year study [3] of 164 consecutive cases of chronic secretory otitis media in 85% of

Read before the Section on Laryngology, Otology, and Rhinology at the One Hundredth Annual Session of the American Medical Association, Atlantic City, June 13, 1951.

1. Williams, H. L.: A Phylogenetic Concept of Allergy, Proc. Staff Meet., Mayo Clin. **24**:516-524 (Sept. 28) 1949.

2. Forman, J.: International Correspondence Society of Allergists, Ser. 14, pp. 1-8, not dated.

3. Jordan, R. E.: Deafness Due to Allergy, Laryngoscope **60**:152-160 (Feb.) 1950.

which the condition was allergic. Chronic secretory otitis media is one of the most important problems in otology because it is usually difficult to diagnose and its complications are often irreversible. Politzer [4] first reported this condition in 1867 under the title "Diagnosis and Treatment of Collection of Serous Fluid in the Middle Ear." After Politzer's contribution very little information on this subject appeared in the literature until 1943, when Hoople and Blaisdale [5] reported 804 cases. There is still a great difference in the numbers of cases observed by various otologists throughout the country. The diagnosis is aided by the characteristic physical appearance of the tympanic membrane and the middle ear. The reflection of the fluid through the drum membrane gives it a reddish, amber color. In contrast to early suppurative middle ear disease, there is little or no dilatation of the tympanic vessels. Air bubbles are rarely seen initially but are often visible after tubal inflation. The tympanic membrane is fixed in a slightly retracted position. It is imperative that a pneumatic otoscope be used to determine the mobility of the membrane. A positive means of diagnosis is a simple paracentesis, followed by tubal inflation and spot suction. In adults this procedure can be accomplished in the office. In children anesthesia is often required. The hearing loss is of a conductive type and averages between 20 and 30 db. in the speech frequencies. The following complications have been observed in untreated or inadequately treated patients with chronic secretory otitis media: irreversible hearing loss resulting from middle ear adhesions involving the tympanic membrane and ossicles, and rupture of Shrapnell's membrane followed by attic suppuration and cholesteatoma resulting from repeated attacks of secretory otitis media. The latter phenomenon has been observed in six of our patients, two of whom required radical mastoidectomy. This complication, previously reported,[3] offers definite clinical evidence that attic cholesteatoma can be a sequela of chronic secretory otitis media. It is generally agreed that closure of the Eustachian tube can result in the appearance of fluid in the middle ear. There is clinical evidence that in such conditions as chronic secretory otitis media the mucosa of the Eustachian tube, middle ear, nose, and nasopharynx may react to allergic stimuli in a similar manner. Ashley,[6] Dean,[7] Jones,[8] Koch,[9] and others have made this observation. Since the mucosa of the Eustachian tube and middle ear may react to allergic stimuli, treatment of the underlying allergy is necessary for effective control and prevention of complications.

Other middle ear disorders can be explained on the basis of the allergic reaction of the mucosa. In 1950, observations on 61 cases of chronic suppurative otitis media with central perforations were made. In 27 of these the condition was compli-

4. Politzer, A.: Diseases of the Ear, edited by M. J. Ballin, Ed. 6, Philadelphia, Lea & Febiger, 1926.

5. Hoople, G. D., and Blaisdale, J. H.: The Problem of Acute Catarrhal Otitis Media, Ann. Otol. Rhin. & Laryng. **52**:359-363 (June) 1943.

6. Ashley, R. E.: Medical Care and Prophylaxis in Hearing Losses with Special Attention to Allergies, Tr. Am. Otol. Soc. **37**:169-177, 1949.

7. Dean, L. W.; Agar, J. S., and Linton, L. D.: Allergic Diseases of the Ear, Laryngoscope **47**:707-728 (Oct.) 1937.

8. Jones, M. F.: Manifestations of Allergy in the Ear, Ann. Otol. Rhin. & Laryng. **47**:910-916 (Dec.) 1938.

9. Koch, H.: Allergical Investigation of Chronic Otitis, Acta oto-laryng., Supp. 62, pp. 1-201, 1947.

cated by allergy. In this group an intermittent profuse serosanguineous discharge from the involved ear was a characteristic finding. Three patients had a profuse aural discharge following the ingestion of food to which they were sensitive. In 1943, Dohlman [10] stated that in many cases of chronic suppurative otitis media in which the ear did not become dry with the usual types of treatment the condition was of allergic character. Later, Koch,[9] in a study of 222 such cases, was able to demonstrate by aural cytology and biopsy the presence of middle ear allergy in 41 cases.

In patients with radical mastoidectomies in which the middle ear mucosa was not completely removed or the Eustachian tube remained open, continued postoperative discharge may be due to allergic reaction. The moisture from the remaining mucosa causes maceration of the skin lining, which delays healing and promotes prolonged discharge. A review of 89 consecutive radical mastoidectomies over a period of four years revealed 17 cases with persistent moist cavities. In ten of these 17 cases the condition was found to be allergic. In all but three of these cases the cavity became dry after allergic management.

During the first six weeks after fenestration an exacerbation of an existing allergy developed in six patients. Simultaneously a slit-like opening appeared along the anteriormost edge of the flap, through which a mucous or serous fluid discharged into the operative cavity. Allergic management was instituted for all the patients. In three patients, the ear became dry after an average of 15 months. The remaining patients continued to have wet cavities intermittently, one patient for six years. This condition did not occur in 24 patients who underwent fenestration, in whom the presence of allergy was recognized prior to the operation and some allergic management instituted.

CONDITIONS INVOLVING THE INNER EAR

The concept that Ménière's disease is caused by a spastic-atonic state of the stria vascularis has focused more attention on allergy of the inner ear. Recent observations by Day [11] have shown that in a majority of cases of Ménière's disease the deafness precedes the vertigo. If the diagnosis can be made prior to the onset of vertigo, treatment is more likely to be effective and the restoration of hearing more frequent. After the onset of vertigo, restoration of hearing by treatment is less frequent. Two important diagnostic symptoms, distortion of sound and hypersensitivity to loud sounds, have been added to the Ménière triad of deafness, tinnitus, and vertigo. These additional symptoms aid in the diagnosis of Ménière's disease prior to the onset of vertigo. The characteristic distortion of sound causes a loss of discrimination out of proportion to the pure-tone hearing loss. Ménière's disease was diagnosed in 51 patients seen in 1950. In 16 patients, the condition was diagnosed before the onset of vertigo.

Fifteen patients with varying types of tinnitus without hearing loss or vertigo were observed in 1950. In five of them the tinnitus ceased after allergic management.

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10. Dohlman, F. G.: Allergiska processer i mellanörat, Nord. med. **20**:2231, 1943.

11. Day, K. M.: Ménière's Disease: Present Concepts of Diagnosis and Management, Ann. Otol. Rhin. & Laryng. **59**:966-979 (Dec.) 1950.

TREATMENT

Of the 727 patients seen during 1950, there were 200, or 27%, with otological conditions caused or complicated by allergy. Although space does not permit a detailed analysis of the methods used to treat these allergic patients, a few suggestions of a general nature are offered.

Treatment cannot be based on skin testing alone. It is essential that all factors included under the broad concept of allergy be considered. The entire allergic constitution of the patient must be investigated.

The antihistaminic drugs are very useful in the management of many allergic patients. Unfortunately, after prolonged continued use these drugs tend to lose their effectiveness. Their use is justified, however, since they prove the diagnosis and afford prompt symptomatic relief during the initial phase of allergic investigation. Sensitivity to certain foods, especially milk, is frequently found to be the etiological factor in chronic secretory otitis media and a complicating factor in

Summary of Clinical Data

Name of Condition or Disease	No. of New Cases Seen in 1950	No. of Cases Caused or Complicated by Allergy	Per Cent of Cases of Allergic Origin	Per Cent of Total No. of Cases Seen in 1950 That Were of Allergic Origin
Chronic otitis externa	47	8	17.0	1.1
Chronic secretory otitis media	111	97	87.4	13.3
Chronic suppurative otitis media with central perforation	61	27	44.2	3.7
Postoperative radical mastoidectomy	35	12	34.3	1.7
Ménière's disease with and without vertigo	51	51	100 *	7.0
Tinnitus without vertigo or deafness	15	5	33.0	0.7
Other cases	407	..	....	....
Total	727	200	....	27.5

* This figure is based on the concept that true Ménière's disease is an allergic phenomenon.

simple otitis media. Emotional disturbances occasionally complicate otological conditions. As an example, an intermittent profuse discharge from simple otitis media in a young nervous mother was repeatedly controlled with phenobarbital. Endocrine dysfunctions, especially thyroid deficiencies, have been encountered in conditions such as chronic secretory otitis media and chronic otitis externa in adult patients.

In Ménière's disease or allergy of the inner ear, the antihistamines have not been effective, although some transitory benefit has been observed in early cases without vertigo. Methantheline (banthine®) bromide (50 to 100 mg. every six hours) has been effective for temporary symptomatic relief. The most consistent, long-term control in our experience has been obtained from weekly or biweekly subcutaneous injections of minimal doses of histamine base.

In order to obtain satisfactory otological and allergic care for these patients, it was necessary to have the allergist work with us in our office. Such an arrangement insures proper observation by physicians of both specialties. It is my opinion that if close cooperation cannot be maintained between members of these two specialties, the otologist should prepare himself to do allergic investigation and treatment.

ABSTRACT OF DISCUSSION

DR. ERHART RUEDEMANN, Pittsburgh: Dr. Jordan has added an entirely new chapter to the ever-widening study of otolaryngologic allergy.

He has described very accurately one of the major types of otological allergy, namely, the hypersecretory ear. Time has not permitted Dr. Jordan to go into the many minor phases of this ear condition. One of these is simple Eustachian tube closure. Usually the tube will open by itself as soon as the active phase of the allergy passes. It is much simpler and more effective to solve this ear problem by an allergic survey and treatment than by repeated tubal inflations.

Polyps in the middle ear are another rare but troublesome problem. These polyps, I am sure, have the same allergic background as have nasal polyps. One must expect a recurrence of the polyps after removal unless the allergic background is investigated and corrected. It has also been my observation that persons who fly a great deal and who repeatedly complain of tubal closure when they fly almost invariably have allergies. Dr. Day and Dr. Jordan have repeatedly warned about fluid in the middle ears of infants after improper feeding. This is especially true of bottle-fed babies. It is believed that entry of milk into the middle ear during feeding, time and time again, creates a shock tissue in this area which later develops into the hypersecretory condition.

Repeated middle ear disease has long been a major indication for removal of tonsils and adenoids. Now much of middle ear disease in children can be shown to be an allergy rather than an infection; therefore the physician must be more careful in his selection of patients for removal of tonsils and adenoids. In children with a conductive hearing defect, it has been common practice to use x-ray or radium treatment in the belief that the defect was always due to hypertrophy of the lymph tissue around the tubal opening brought on by chronic infection. Now much of this hypertrophy is known to be caused by allergy. In fact, instead of hypertrophy, there may even be allergic edema, so that one must exercise more discretion in the use of x-rays or radium for treatment of this type of hearing defect. Dr. Jordan is right when he says that the otolaryngologist should associate himself closely with the allergist if he does not do his own allergic investigation. I shall go a step further and say that the otolaryngologist must be his own allergist. It is no great task to make an allergist from an otolaryngologist, but one can never make an otolaryngologist from an allergist.

DR. F. W. DAVISON, Danville, Pa.: Dr. Jordan has, in a very convincing manner, called our attention to the fact that an allergic reaction can be a factor in many cases of disease of the external, middle, or inner ear. I think our understanding of these cases would be clarified if each time we used the word allergy we would specify the type of allergy existing in the patient under discussion.

H. L. Williams has classified allergy into three types: Type 1, physical allergy; Type 2, tissue or bacterial allergy, and Type 3, humoral or circulating antibody allergy. In addition, there are many disease states due to dysfunction of the autonomic nervous system which present a clinical appearance almost indistinguishable from the three types of allergy just mentioned. I do not think the word allergy should be applied to the disease states resulting from autonomic imbalance. I think that Hilger has selected appropriate terms in the designations vasomotor labyrinthitis, vasomotor vestibulitis, and vasomotor cochleitis. For a long time vasomotor rhinitis has been a descriptive and understandable diagnostic label properly applied to nasal disturbance resulting from autonomic dysfunction to distinguish it from symptomatically identical nasal disturbances due to antigen-antibody reaction resulting from either food or inhalant sensitivity. Arteriolar spasm is the common denominator present in each condition, but if the patient is to receive proper therapy the factor responsible for the vasospasm must be determined and, if possible, corrected. The fact that severe vasospasm is caused by the antigen-antibody type of allergy was beautifully demonstrated by the work of Abell and Schenck. They showed that arteriolar spasm occurred in the blood vessels of a sensitized rabbit's ear when the antigen was topically applied, as well as when the antigen was carried by the circulation.

Some persons inherit a labile autonomic system and others a stable nervous system. It so happens that the person who inherits the tendency to develop the antigen-antibody type of allergy also inherits a labile autonomic system. As pointed out by Draper, Dupertuis, and

Caughey in their book "Human Constitution in Clinical Medicine," "No person with stable autonomic function ever can fully appreciate the uncertainty and anxiety which the labile autonomic equipment brings to its owner."

I agree with Dr. Jordan that secretory otitis media is a very frequent, and also a frequently overlooked, condition, but I do not find allergy—at least the antibody type of allergy—to be an etiological factor in as high a percentage of cases as he reports. In a series of 40 case histories which I recently reviewed, the antigen-antibody type of allergy was apparently an etiological factor in 43%. In my opinion there is no excuse for use of a radium applicator in the nasopharynx if the patients for whom its use has been suggested are treated with a careful, clean, surgical adenoidectomy, proper management of any existing allergy, and correction of faulty dietary habits—the most frequent of which is a high carbohydrate diet.

I cannot agree with Dr. Jordan that the antigen-antibody type of allergy is an etiological factor in all cases of Ménière's disease. In my experience, Ménière's disease is much more frequently due to vasospasm resulting from emotional stress than to vasospasm resulting from antigen-antibody allergy. I think it is a mistake to class vasomotor labyrinthitis as an allergy. As Dr. Jordan stated, the antihistaminic drugs have not been effective in a large percentage of cases of Ménière's disease. This suggests that the disease is not frequently due to the antigen-antibody type of allergy.

I certainly agree with Dr. Jordan that the otologist should make his own allergy studies and that the study of allergy in the broadest sense of the word should be a major subject in the postgraduate training of otolaryngologists. Dysfunctions of the autonomic nervous system should also receive considerable attention, but they should not be labeled allergies simply because persons in whom the antigen-antibody type of allergy develops are, in addition, apt to have an unstable autonomic nervous system.

DR. R. E. JORDAN, Pittsburgh: I should like to make a few remarks concerning Dr. Davison's discussion. Every time that a paper is given on allergy, of course, the same question comes up: What is allergy? Obviously, in a 15-minute paper, no one can clearly define what is meant by allergy. I attempted in the opening paragraph to convey the fact—perhaps I did it badly—that I do not believe that all allergy is an antigen-antibody reaction by any means, that I include all the autonomic dysfunctions, psychic changes, endocrine dysfunctions, and so on, under the broad term, allergy.

I believe Dr. Davison's term, "the allergic constitution," is one of the finest contributions to this muddled definition of allergy that we now know today. I don't believe allergy is a nose or an ear; it is a complete individual, and that individual may show reactions of what we call an allergic nature to many stresses and strains, as Forman so admirably put it. In Ménière's disease I believe that there is rarely an antigen-antibody reaction. I can think of only one case in our series in which we could attribute the patient's condition to a reaction to dust sensitivity. We treated the patients having Ménière's disease with histamine, and we believe that the response is favorable; but I have yet to observe a case in which I thought Ménière's disease was due to a food of a specific nature or to a specific allergen. Histamine helps to stabilize the autonomic nervous system, and thus is helpful in the control of allergic reactions.

PATHOLOGY OF OTOSCLEROSIS

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THE RESULTS of surgical treatment of otosclerosis are certainly more fascinating than the practical results obtained from studies on the pathology of otosclerosis. In my opinion, conclusions which have been drawn from microscopic findings are disappointing from the practical point of view. Nevertheless, these studies must be continued because the final solution of the problem of otosclerosis must result from pathologic studies and not from surgery. For this reason I feel that it is timely to summarize the present status of the pathology of otosclerosis. This paper does not aim at a presentation of the bibliography, which has been much better accomplished in a résumé of the literature by the Central Bureau of Research of the American Otological Society, Inc., but rather gives my personal views based mainly on work covering the past 25 years. This presentation is permissible because my concept, presented in 1924,[1] has to a certain extent stood the test of time. It must be admitted that this concept does not furnish more practical results than others. The aim is rather to classify the various microscopic findings in otosclerosis and to formulate precisely the questions which may be answered at a future time.

Otosclerosis is a primary disease of the bony capsule of the labyrinth, that is, it originates within the bone and not within the tympanic mucosa. This statement, made by Politzer in 1894,[2] is generally accepted. In 1924 I classified the microscopic findings in otosclerosis according to those changes which create a "disposition" to otosclerosis and those of actual otosclerosis. Several investigators have accepted this classification although they did not have in mind the same changes. At present I use the terms "preotosclerotic" and "otosclerotic" changes. These are analogous to the terms "precancerous" and "cancerous" changes.

PREOTOSCLEROTIC FINDINGS

The following changes I have called "preotosclerotic findings": (*a*) degeneration of the cartilage at the frame of the windows,[3] and (*b*) the "blue mantles" of the blood vessels.

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1. Brunner, H.: Beiträge zur Histogenese des otosklerotischen Knochens, Ztschr. Hals-, Nasen- u. Ohrenh. **6**:320, 1923; Beiträge zur Pathologie des knöchernen Innenohres mit besonderer Berücksichtigung der Otosklerose, Monatsschr. Ohrenh. **58**:1-62, 1924.

2. Politzer, A.: Über primäre Erkrankung der knöchernen Labyrinthkapsel, Ztschr. Ohrenh. **25**:309, 1894.

3. The cartilage at the frame of the windows and within the petrous bone is not hyaline cartilage but chondroid supporting tissue (Schaffer, Brunner) which is found only in a few places in the human body.

(*a*) The degeneration of cartilage consists in a loss of the nuclei and a deposition of calcium salts. I have noticed the degeneration in instances of actual otosclerosis as well as in instances without otosclerosis. For this reason the findings were called preotosclerotic. Degenerative changes are by no means unusual in normal cartilage. In fact, "fading cells" can be discovered in almost every specimen of cartilage. These are cells which lose their capsule and their nucleus and disappear in the ground substance. In preotosclerotic petrous bones the same changes are present but are very much increased in intensity and extensity. The degeneration of the cartilage at the frame of the windows is not otosclerosis but is a preotosclerotic finding. This fact is important because several otologists claim that actual otosclerosis is preceded by a "degeneration" of the original bone of the labyrinth capsule. I am opposed to this concept as far as the bone is concerned. However, degenerative changes of the cartilage at the frame of the windows may, and frequently do, precede actual otosclerosis. It is not known whether the cartilage of the interglobular spaces undergoes similar changes.

(*b*) The "blue mantles" of the blood vessels have been described by Manasse.[4] The term "mantle" is a misnomer because a mantle covers the body loosely while the blue mantles adhere snugly to the walls of the blood vessels. Actually the blue mantles are to be found in every normal temporal bone. However, in normal circumstances they form very narrow sheaths, and for this reason they are called marginal sheaths of the blood vessels. In the preotosclerotic stage the marginal sheaths are broadened and may eventually cause an obliteration of the blood vessel. The broadened blood vessels sheaths are the blue mantles of Manasse. They consist of a substance which is strictly basophilic and stains intensely with hematoxylin, does not contain cells or fibrils, and is produced by the endothelial cells of the capillaries. This substance is not keratin and is, primarily, not bone because it does not contain fibrils. For this reason, von Ebner and Schaffer [5] have called it "precollagenous substance" [5a] because, in contrast to collagen, it does not turn to glue or to gelatin when boiled.

The blue mantles usually change into genuine bone substance with the appearance of cells and fibrils. This is due to the expansion of the precollagenous substance, to the activity of ferments and, particularly, to mechanical forces exerted by the growing tissue on the precollagenous substance. The newly formed bone differs markedly from the normal lamellar bone in the following respects: 1. The cells are irregularly scattered throughout the newly formed bone and are lacking in canaliculi. They frequently are encased in dilated cavities. The cells of the lamellar bone form regular rows and have an intricate system of canaliculi. 2. The fibrils of the newly formed bone are sparse and form an irregular network. The fibrils of the lamellar bone are abundant and form regular lamellae. 3. The newly formed bone stains with hematoxylin, the intensity of staining being in inverse proportion to the content of fibrils. The lamellar bone is distinctly eosinophilic. 4. The newly

4. Manasse, P.: Neue Untersuchungen zur Otosklerosenfrage, Ztschr. Ohrenh. **82**:76, 1922.

5. Schaffer, J.: Vorlesungen über Histologie und Histogenese, Leipzig, Wilhelm Engelmann, 1920.

5a. A more exhaustive discussion of the precollagenous substance can be found in articles by Zawisch-Ossenitz (Ztschr. mikrosk.-anat. Forsch. **6**:76, 1926; **18**:393, 1929; **42**:595, 1937) and in that by Landauer (J. Biol. Chem. **108**:121, 1935).

formed bone is not deposited on the islands of cartilage like the lamellar bone; it rather shows a distinct relationship to the blood vessels.

These findings prove that the newly formed bone is a primitive one which is called "woven bone" or "web(like) bone." In advanced cases the web-like bone is formed not only by a simple transformation of precollagenous substance but, in addition, by a genuine ossification of connective tissue in the labyrinth capsule. This concept differs only in one point from the viewpoint of other otologists. Several claim that the blue primitive bone develops exclusively by ossification of connective tissue subsequent to resorption of original bone of the labyrinth capsule, I simply add the theory that the primitive blue bone may also develop from a transformation of precollagenous substance without preceding resorption of bone and without ossification of connective tissue. In any case, it seems to be an established fact that the blue primitive bone is newly formed and is not original bone in a state of degeneration.[6]

The "blue mantles" as well as the blue primitive bone are not normal findings, but they are frequent in otosclerosis, in which they are found in both the otosclerotic focus and the rest of the petrous bone, outside of the otosclerotic focus. Weber[7] found them in 58 instances among 82 cases of otosclerosis. Since they are absent or infrequent in other diseases of the labyrinth capsule, there must be a relationship between otosclerosis and the blue mantles.[8] However, the latter are not evidence of otosclerosis for the following reasons: 1. They are diffusely distributed in the labyrinth capsule, that is, they are to be found in places where actual otosclerosis is rare, as in the area of the vertical semicircular canals. In contrast, actual otosclerosis is a localized disease. 2. They may be noticed in petrous bones where no actual otosclerosis is to be found. This point was emphasized by me in 1924. Weber[7] discovered blue mantles in 19 instances (25%) among 77 patients who had no actual otosclerosis. In summary, it becomes evident that neither the blue mantles nor the blue primitive bone per se constitutes otosclerosis but that both should be classified as preotosclerotic changes inasmuch as they do not cause clinical symptoms. This is contrary to the view of Mayer,[6] who apparently considers that the blue mantles constitute otosclerosis.

The frequent association of actual otosclerosis with preotosclerotic changes may indicate that the former develops exclusively in petrous bones which harbor preotosclerotic changes. At present, this statement is premature. However, if it were correct, the term "preotosclerotic" would become more precise than the term "precancerous" because actual carcinoma may and frequently does develop without being preceded by precancerous changes.

This concept concerning the preotosclerotic changes, particularly the blue mantles of Manasse, was presented by me in 1924 and was confirmed by the

6. Mayer, O.: Untersuchungen über die Otosklerose, Vienna, Alfred Hölder, 1917.

7. Weber, M. H. E.: Otosklerose und Umbau der Labyrinthkapsel, Leipzig, Poeschel & Trepte, 1935.

8. Exostoses and malformations of the internal ear may be likewise associated with otosclerosis, but by no means as frequently as the "blue mantles." For this reason these findings should not be classified as preotosclerotic changes.

extensive studies of Nager and Meyer[9] and of Weber,[7] although the latter employed a somewhat different terminology.[10]

FINDINGS IN OTOSCLEROSIS

Onset of the Disease.—There are essentially two views which have been advanced, as follows: 1. At the onset of actual otosclerosis the original bone of the labyrinth capsule is resorbed by osteoclasts and the defects are filled with newly formed bone which, however, differs in its structure from the original bone. 2. At the onset of actual otosclerosis there is a "degeneration" of the original bone of the labyrinth capsule and the degenerated bone gives rise to the action of osteoclasts and osteoblasts.

The chief exponent of the first view is Mayer[6] and of the second view, Wittmaack.[11] I have always shared the first view. If at the onset of the disease there would be a "degeneration" of the original bone, one should expect the same degeneration, close to a growing otosclerotic focus. However, there is no degeneration of the bone surrounding a growing otosclerotic focus. In addition, the modern methods of silver staining and examination with polarized light (Weber,[7] Nager and Meyer[9]) have definitely proved the correctness of the first view, and, thus, Weber[7] summarizes that the entire bone of an otosclerotic focus is newly formed, subsequent to a resorption of the original bone by osteoclasts. In other words, there is no "degenerated" bone in an otosclerotic focus.

It should be recalled that what is correct for the bone is not necessarily correct for the cartilage at the frame of the windows. In fact, I hold the opinion that there is an atrophic degeneration of the cartilage which precedes actual otosclerosis, as mentioned before.

Resorption of original bone being the initial stage of actual otosclerosis, it is evident that the diagnosis of preotosclerotic changes must be changed to the diagnosis of actual otosclerosis if in the area of preotosclerotic changes there is a definite resorption of bone by osteoclasts or definite apposition of bone by osteoblasts, subsequent to resorption of bone. This is the onset of actual otosclerosis, which is invariably a localized disease favoring certain areas of the petrous bone, in contrast to the preotosclerotic changes, which extend all over the petrous bone. Frequently the otosclerotic foci are multiple. However, there is no answer to the question of why otosclerotic foci favor certain spots of the petrous bone.

Course of the Disease.—When the otosclerotic focus is formed, it may be active or inactive. A final stage does not exist because the focus may regain activity even in the old age of the patient. If the focus is inactive, there is no, or very slight, osteogenesis and practically no osteoclasis. The otosclerotic bone, which is invariably a woven bone, becomes gradually atrophic. The cell nuclei disappear, and there are empty cavities and deep lacunae in the bone. The

9. Nager, F. R., and Meyer, M.: Erkrankungen des Knochensystems der Labyrinthkapsel, Berlin, S. Karger, 1932.

10. Weber[7] has noticed that in actual otosclerosis the enchondral layer of the labyrinth capsule, outside the otosclerotic focus, shows a transformation proceeding according to the normal pattern, which, however, exceeds the normal findings in intensity and extensity. I do not consider this finding to be a preotosclerotic change because it is not clear whether it precedes actual otosclerosis or is rather the reaction to an actual otosclerotic focus. Disregarding this question, I can confirm the finding.

11. Wittmaack, K.: Die Otosklerose, auf Grund eigener Forschungen, Jena, Gustav Fischer, 1919.

atrophy is most marked where the focus abuts on the connective tissue of the endosteum or the fissula ante fenestram or the spiral ligament of the cochlea.

If the focus is active, the newly formed bone is resorbed and other bone is deposited which usually is again web-like and exceptionally lamellar bone.[12] This process represents the internal growth of the otosclerotic focus. But there may be, in addition, an external growth, viz., an extension of the focus. The focus grows in the same manner as it originates, viz., the original bone of the labyrinth capsule is resorbed and new bone fills up the defect. The focus grows within the bone exclusively, particularly within the enchondral layer of the labyrinth capsule. Connective tissue is a relative impediment to the growing focus. The bone prefers to grow around the connective tissue and may even become atrophic where it abuts on connective tissue. Finally, of course, the connective tissue gives way and the bone grows through. For this reason, the connective tissue of the fissula ante fenestram is not infrequently noticed in the center of the otosclerotic focus; the annular ligament may be spared although there is a large focus at the frame of the window. Most resistant is the endosteum of the internal ear. The otosclerotic focus may bulge the endosteum slightly into the scala tympani, but it does not grow through the endosteum to fill up the perilymphatic space. On the contrary, the otosclerotic bone close to the endosteum frequently becomes atrophic.

There is only one exception. If the scala tympani is filled with newly formed bone subsequent to an infection or an injury to the internal ear, the otosclerotic, primitive bone may invade the newly formed bone within the inner ear, a finding which again proves the tendency of the otosclerotic bone to grow within bone. In very exceptional cases otosclerosis extends to the dura at the surface of the petrous bone.

If the otosclerotic bone extends toward the mucosa of the tympanic cavity, the latter becomes fibrotic and hyperemic. This causes Schwartze's sign, which, however, does not indicate necessarily activity of the otosclerotic focus, for the tympanic mucosa may be found to be thickened and hyperemic and yet the underlying otosclerotic focus is only slightly, or not at all, active.

While the otosclerotic focus may bulge only slightly into the perilymphatic spaces of the internal ear, it not infrequently causes tumors of considerable size, which particularly bulge into the recessus of the windows and may eventually cause an obliteration of the recessus.

Diffuse Otosclerosis.—In some cases of multiple otosclerotic foci, the foci fuse and almost the entire capsule of the labyrinth presents preotosclerotic and otosclerotic changes. Even in these extreme cases the primitive bone may be kept away from the dura at the surfaces of the petrous bone. Conspicuously, stapes ankylosis may be absent in these cases, but there is not infrequently a labyrinthitis ossificans (Nager and Meyer,[9] Brunner[13]).

CLASSIFICATION OF OTOSCLEROSIS

If an attempt is made to classify otosclerosis as an entity, the distinction between preotosclerotic and otosclerotic changes must be kept in mind.

12. This, of course, does not imply that there are haversian systems.

13. Brunner, H.: Otosclerosis Associated with Osteoporosis and Labyrinthitis Chronica Ossificans, Arch. Otolaryng. **49**:184 (Feb.) 1949.

Preotosclerotic Changes.—There is very little known about these changes. It is not even known in what age group they are noticed first. I have seen preotosclerotic changes, viz., "blue mantles" and degeneration of the cartilage at the frame of the oval window, in a boy aged 9 years who died from a head injury and had no actual otosclerosis, perhaps because of his young age. Studies about preotosclerotic changes in various periods of life are, to my knowledge, not available. Only this much can be said: In otosclerosis the osteoblasts which derive from the endothelial cells of the capillaries produce precollagenous substance and primitive bone instead of normal lamellar bone. The etiology of this anomaly is not known. One may refer to constitutional anomalies inasmuch as it seems that the preotosclerotic changes are hereditary. However, this theory is conjectural.

Otosclerosis.—Several views have been advanced in the classification of otosclerosis from the standpoint of pathology. The otosclerotic foci have been classified as tumors, callus formation, localized osteitis fibrosa, and bone dystrophy, to mention only the chief concepts. A few of these can be discarded. Neither in human pathology nor in animal experiments have facts been found to support the hypothesis that otosclerosis represents bone dystrophy. Otosclerosis is likewise not a callus formation because there are microscopic fissures at sites of the temporal bone where an otosclerotic focus is never noticed.[14] So far as localized osteitis fibrosa is concerned, I share the opinion of Jaffe and Lichtenstein,[15] who claim that "localized osteitis fibrosa is a waste-basket term in bone pathology, holding many ill-assorted concepts" and who state that they know of no sensible answer to the question: What is localized osteitis fibrosa?

The idea that the otosclerotic focus is a tumor or a tumor-like formation has been advanced by several authors. Mayer [6] has particularly favored this concept. He called the otosclerotic foci "tumor-like hyperplasias belonging to the hamartomas," [16] and he emphasized the following facts in support of this idea: (1) the distinct margins of the foci; (2) the finding of primitive bone within the foci; (3) the frequent formation of hyperostoses which bulge into the tympanic cavity and infrequently into the internal ear. Mayer, however, does not speak of tumors but of tumor-like formations because the foci do not grow by expansion according to the pattern of benign tumors. In another paper [17] Mayer made an attempt to fuse this concept with his idea that the otosclerotic focus is a callus formation, and thus he obscured both concepts. I have approached the problem from another angle. A great deal of work has been done in interpreting the histologic features of the otosclerotic focus, but the biology of the focus as a whole has been neglected. It is just this aspect which attracted my attention. From these studies the following conclusions can be drawn:

1. When the otosclerotic nidus grows, it does so autonomously, viz., the growth, internal and external, is entirely independent of the growth of the rest of the labyrinth capsule. After the second year of life there is no external and

14. Brunner, H.: Über Spaltbildung in der knöchernen Innenohrkapsel, Wien. klin. Wchnschr. **44**:173-176, 1931.

15. Jaffe, H. L., and Lichtenstein, J.: Osteoid Osteoma: Further Experience with This Benign Tumor of Bone, J. Bone & Joint Surg. **22**:645, 1940.

16. Hamartomas represent an anomalous development.

17. Mayer, O.: Referat über die pathologische Anatomie und Pathogenese der Otosklerose, Deuxième Congrès international d' oto-rhinolaryngologie, Madrid, 1932, p. 7.

almost no internal growth of the normal enchondral layer of the labyrinth (Mayer [6]), and yet the otosclerotic nidus, which involves particularly the enchondral layer, shows usually an internal growth and frequently an external growth, the growth being atypical. For this reason, the growth of the otosclerotic nidus must be called autonomous.

2. Mayer [6] has noticed that in petrous bones which were afflicted by both otosclerosis and osteoporosis the microscopic findings of the otosclerotic nidus were the same as those in cases of otosclerosis without osteoporosis. This statement, with minor modifications, was confirmed by me.[13] In other words, the otosclerotic nidus is not affected by the changes taking place in the rest of the labyrinth capsule.

This autonomy of the otosclerotic focus became more evident in a case of Gaucher's disease [18] and in a case of ochronosis.[19] In Gaucher's disease the cerebroside kerasin or a closely related cerebroglucoside circulates in the blood and is deposited in the cells of Gaucher, notably in the liver, spleen, lymph nodes, and bone marrow. In a case of Gaucher's disease the Gaucher cells have been discovered in the marrow spaces of the petrous bone. There was, in addition, a large otosclerotic focus in the area of the windows. The focus contained a great number of marrow spaces; yet it was not possible to discover definite Gaucher cells in the otosclerotic focus although these cells were noticed in the marrow spaces of the labyrinth capsule, close to the otosclerotic focus. This indicates that the metabolism of the otosclerotic focus differs from the metabolism of the rest of the labyrinth capsule, that is, that the huge otosclerotic focus has been autonomous as far as metabolism and growth are concerned.

In ochronosis homogentisic acid circulates in the blood. Because of chemical or fermentative action, a pigment is formed from the homogentisic acid, and this pigment is deposited in the cells of the cartilage. The petrous bone differs from the rest of the skeleton because in it the pigment is not deposited in the cartilage but in the periosteal and endosteal layer of the bony capsule. In the enchondral layer only a few osteocytes contain the pigment. This fact is self-evident; the enchondral layer contains only a few patent blood vessels and has for this reason a markedly diminished metabolism. However, in the case which I observed there was an otosclerotic focus in the window area, in addition to the ochronosis. The focus was well vascularized and yet there was no pigment present in it, although there was a great amount of pigment in the periosteal bone, close to the focus. This is a crucial experiment, made in natural circumstances and proving that the metabolism of the otosclerotic focus is autonomous, in other words, that in the otosclerotic focus there is a new metabolism which is not normal and is not subject to the normal restraints.

All these findings prove that growth and metabolism of the otosclerotic focus proceed autonomously. Autonomy of growth and metabolism is the fundamental feature of a tumor. Ewing [20] expresses this idea most precisely by saying that a

18. Brunner, H.: Über das Vorkommen von Gaucherzellen im Felsenbein nebst Bemerkungen zur kausalen Genese der Otosklerose, Ztschr. Hals-, Nasen- u. Ohrenh. **22**:60, 1928.

19. Brunner, H.: Über die Veränderungen des Schläfebeines bei der Ochronose, Monatsschr. Ohrenh. **63**:997, 1929.

20. Ewing, J.: Neoplastic Diseases, Ed. 2, Philadelphia, W. B. Saunders Company, 1928.

neoplasm or tumor is an autonomous new growth of tissue. If, in addition, the findings which Mayer [6] has emphasized in favor of the tumor-like nature of the otosclerotic focus are recalled, it is evident that otosclerotic foci are not tumor-like formations but genuine tumors of the capsule of the labyrinth.

In an attempt to determine the nature of the otosclerotic tumors more precisely, the following facts must be stressed: The otosclerotic tumors are primary tumors of the bony capsule of labyrinth and probably originate in the area of the borderline of the enchondral and periosteal layers. They may be solitary or multiple, and, if multiple, they may fuse. They are frequently symmetrically formed on both sides. They are usually associated with preotosclerotic changes in the rest of the labyrinth capsule. They grow by resorption of the original bone of the labyrinth capsule. They are specific tumors of the capsule of the human labyrinth. Most of these points are self-evident, while some require a comment.

Siebenmann [21] claims that the otosclerotic nidus originates at the borderline of the enchondral and periosteal layers of the labyrinth capsule, while Mayer [6] holds the view that it originates in the periosteal layer. I have seen a very small otosclerotic nidus in a woman, aged 36 years, who died from a cerebellar tumor. The nidus had an atypical localization, namely, it was localized in the bony capsule surrounding the tip of the cochlea where the three layers of the capsule are very distinct. It occupied both the enchondral and the periosteal layer but did not extend either to the endosteum or to the mucosa of the middle ear. This finding proves that the otosclerotic nidus may involve both the enchondral and the periosteal layer in an early stage. Inasmuch as this finding is in agreement with the reports of other authors, it seems that the view of Siebenmann is closer to the facts than that of Mayer.

The otosclerotic tumors grow slowly and intermittently. Sometimes they cease to grow very early. They do not grow by expansion, that is, by pushing aside the surrounding structures, an opinion which has been entertained particularly by Manasse.[4] However, Mayer [6] has definitely proved that they grow by resorbing the original bone and by refilling the defect with otosclerotic primitive bone. In other words, they do not grow like typical osteomas. In this respect the otosclerotic tumors are somewhat unique, but so is their mother tissue, the bone of the capsule of the human labyrinth. This fact, however, does not mean that the otosclerotic tumors grow according to the pattern of malignant tumors because there is no unlimited growth although the tumors may grow into adjacent structures such as the annular ligament, fissula ante fenestram, modiolus, and others.

The peculiar type of growth indicates that the otosclerotic tumors are specific neoplasms of the labyrinth capsule. This is to be expected, since the bone of the capsule of the labyrinth which gives origin to these tumors has a specific structure, different from the bones of the rest of the skeleton. Another finding which confirms the specificity of the otosclerotic tumors is the fact that otosclerotic tumors have been discovered only in the capsule of the human labyrinth. There is only one tumor which resembles otosclerotic tumors, and that is the osteoid osteoma, which is most frequent in the long bones. A comparison between the two types

21. Siebenmann, F.: Über die Anfangsstadien und über die Natur der progressiven Spongiosierung der Labyrinthkapsel (Otospongiosis progressiva), Verhandl. d. deutsch. otol. Gesellsch. **21**:186, 1912.

of tumors proves that the otosclerotic tumors are not simply osteoid osteomas of the labyrinth capsule. However, there are several features which both tumors have in common, while they differ in others. The features which both have in common are as follows:

1. Osteoid osteomas are benign osteogenic tumors of slow growth (Jaffe and Lichtenstein,[22] Ewing,[20] Ponseti and Barta,[23] Sherman,[24] Jackson,[25] and others) and so are the otosclerotic tumors. It is interesting that, like otosclerotic tumors, osteoid osteomas have been considered to be callus formations or inflammatory lesions, views which have been controverted by clinical and pathological facts in both.

2. Osteoid osteoma originates in the cortex or in the marrow of the long bones favoring the area of the borderline between cortex and marrow. Otosclerosis likewise favors the area of the borderline between two layers of bone which are different, structurally and biologically, viz., the periosteal and enchondral layers.

3. At the site in the cortex where osteoid osteoma is developing, intense reconstruction and vascularization of the original cortical bone set in. The original cortical bone at the site is soon substantially broken up and resorbed but is replaced by highly atypical osseous tissue deposited by the osteogenic tissue carried on the walls of the blood vessels which are invading the area (Jaffe[26]). This is exactly what happens in otosclerosis.

4. When the osteoid osteoma develops in small bones consisting mainly of spongiosa it abuts on, and even erodes, the overlying cortical shell; it may elevate the periosteum, but it does not penetrate the latter (Jaffe and Lichtenstein[22]). Otosclerosis may likewise elevate the periosteum or the endosteum, but it does not penetrate either one.

The two tumors differ in the following features:

1. Both otosclerosis and osteoid osteoma develop without apparent cause. However, in osteoid osteoma there are no findings analogous to the preotosclerotic findings and thus there is no hereditary factor in osteoid osteoma.

2. Otosclerosis consists in solitary or multiple tumors, and the tumors are frequently symmetrically formed on both sides. Osteoid osteoma is apparently a solitary tumor and not symmetrical on both sides.

3. Osteoid osteoma is a small, self-limited lesion. In its evolution it follows a consistent pattern. It passes from (*a*) an initial stage in which actively proliferating and compacted osteoblasts may be prominent through (*b*) an intermediate phase in which osteoid in various stages of calcification is conspicuous to (*c*) the mature stage in which the lesion resembles an osteoma composed of densified trabeculae of highly calcified atypical bone (Jaffe and Lichtenstein,[22] Sherman,[24] Jackson[25]). In this respect osteoid osteoma differs strictly from otosclerosis because otosclerotic tumors do not follow a consistent pattern, nor are they self-

22. Jaffe, H. L., and Lichtenstein, L.: Osteoid Osteoma: Further Experience with This Benign Tumor of Bone, J. Bone & Joint Surg. **22**:645, 1940.

23. Ponseti, J., and Barta, C. K.: Osteoid Osteoma, J. Bone & Joint Surg. **29**:767, 1947.

24. Sherman, M. S.: Osteoid Osteoma, J. Bone & Joint Surg. **29**:918, 1947.

25. Jackson, A. S.: Osteoid Osteoma, Proc. Staff Meet., Mayo Clin. **24**:380, 1949.

26. Jaffe, H. L.: Osteoid-Osteoma of Bone, Radiology **45**:319, 1945.

limited, because they may regain activity at any time. This is a fundamental difference.

4. There is a widespread perifocal zone of bone sclerosis and bone thickening surrounding the central nidus which may increase to form a large mass when the osteoid osteoma originates in the cortex. In otosclerosis there is a moderate reconstruction of the rest of the labyrinth capsule (Weber[7]), but there is no perifocal zone of sclerosis. Since a factor in the explanation of the perifocal sclerosis in osteoid osteoma is apparently the irritation of the bone-forming tissue by the slow, torpid growth of the lesion, the absence of the perifocal sclerosis in otosclerosis is likely to be due to the meager, bone-forming capacity of the human labyrinth capsule. For the same reason the perifocal osteosclerosis is milder when the osteoid osteoma appears in the spongy bone of the marrow and not in the cortex.

5. Osteoid osteoma does not cause a thickening or deformity of the bone although the perifocal osteosclerosis may do so, even to a high degree. Otosclerosis per se may, and frequently does, cause a thickening of the bone.

6. The chief symptom of osteoid osteoma is pain. There is no pain in otosclerosis.

7. Osteoid osteoma occurs in young adults or adolescents. Otosclerosis is found in any age except in childhood, when it is rare.

The comparison proves that there are several features in which otosclerosis and osteoid osteoma differ. Nevertheless, the confrontation of osteoid osteoma and otosclerosis indicates that otosclerosis no longer occupies the strictly isolated position in bone pathology which it has maintained up to the present. It seems rather that otosclerosis is a specific type of osteoma.

OTOSCLEROSIS AND THE INTERNAL EAR

It is common knowledge that in otosclerosis there is not infrequently an atrophy of the vestibular and basilar portions of the cochlea. This is a simple atrophy which does not show specific features. In many instances it is caused by a coincidental disease such as arteriosclerosis, syphilis, or head injuries. However, there is another group of cases in which the history does not reveal any lesion of the inner ear. In these instances a relationship between a lesion of the inner ear and otosclerosis must be assumed, and thus the question arises, "Is the lesion of the inner ear caused by the otosclerotic nidus, or does the stimulus which is acting in otosclerosis cause this additional lesion?"

It was probably Siebenmann[27] who first advanced the opinion that the inner ear lesion is caused by a serous labyrinthitis which in turn is due to metabolites passing from the otosclerotic nidus into the internal ear. In the past I have used this theory to explain the serous labyrinthitis in cases of Paget's disease of the petrous bone. Further experience has proved that this concept is not tenable because the serous labyrinthitis had to be considered as vasomotor internal otitis, which may or may not be associated with Paget's disease. It is likely that the serous labyrinthitis which, infrequently, is associated with otosclerosis has the same pathogenesis.

27. Siebenmann, F.: Totaler knöcherner Verschluss beider Labyrinthfenster und Labyrinthitis serosa infolge progressiver Spongiosierung, Verhandl. d. deutsch. otol. Gesellsch. **20**:267, 1911.

Likewise untenable is the idea of Mayer [6] that the inner ear lesion is caused by a passive hyperemia which in turn is due to an otosclerotic nidus in the modiolus obliterating the veins of the cochlea. There are many cases of otosclerosis which do show an atrophy of the cochlea but no otosclerosis in the modiolus.

These considerations seem to indicate that the inner ear lesion is caused by the same factor which causes the formation of the preotosclerotic changes.[28] If this concept is correct, one must expect to make the following observations: 1. There must be cases of otosclerosis in which deafness is caused by an inner ear lesion but not by stapes ankylosis. 2. In cases of unilateral otosclerosis there must be occasionally an inner ear lesion on the side which is not afflicted by otosclerosis. In regard to the first point, there are clinical cases of otosclerosis on record in which the deafness is, from the onset, the perceptive type and in which Gellé's test indicates a motility of the stapes. However, to my knowledge, there is no microscopic proof that these are actually cases of otosclerosis without stapes ankylosis. In regard to the second point, I have observed two cases which support the concept of the independence of the inner ear lesion from the otosclerotic tumor.

The first case concerned a woman, aged 36, who died from a tumor of the midbrain. There was no history of infection, poisoning, or injuries. The examination revealed a small otosclerotic nidus without stapes ankylosis on the right side, but there was an atrophy of the basilar coil of the cochlea on both sides. In an earlier paper [29] I have assumed that the atrophy of the cochlea was caused by a degeneration descending from the quadrigeminal body down to the spiral ganglion in the cochlea. At present, it seems more plausible that the atrophy of the cochlea was caused by the same factor which was acting in the formation of the preotosclerotic changes.

In the second case a man, aged 25, died from a cerebellar abscess. There was an otosclerotic nidus in front of the right oval window. On the left side there was no otosclerosis, but there was an atrophy of the basilar portion of the cochlea, a degeneration of the cartilage at the frame of the oval window, and a large marrow space at the insertion of the membrane of the round window containing a blood vessel and osteoclasts; there were no "blue mantles."

SUMMARY

In otosclerosis there are preotosclerotic and otosclerotic changes. The preotosclerotic changes consist of (1) an overproduction of both precollagenous substance and primitive bone and (2) an extensive degeneration of the cartilage at the frame of the windows. These changes are probably hereditary and do not produce clinical symptoms. They are called "preotosclerotic" because (*a*) they are frequently associated with actual otosclerosis, (*b*) they occur also in instances without actual otosclerosis, (*c*) they are not associated with other diseases of the bone of the labyrinth, and (*d*) they are, as far as the "blue mantles" are concerned, distributed all over the petrous bone.

28. Since actual otosclerosis is a genuine tumor, it cannot be assumed that the (unknown) factor which causes the formation of a tumor of the bone should also cause an atrophy of Corti's organ and its nerve.

29. Brunner, H.: Otologische Diagnostik der Hirntumoren, Berlin, Urban & Schwanrzenberg, 1936.

The preotosclerotic changes turn to actual otosclerosis if there are signs of activity, viz., definite osteoclasis and/or osteogenesis in these areas.

The actual otosclerotic nidus is a genuine tumor of the bone of the labyrinth capsule which presents various microscopic features according to the stage of activity. As in all tumors, the beginning of the disease is scarcely demonstrable.

The otosclerotic tumors have certain features in common with osteoid osteoma. However, they are not osteoid osteomas, differing from the latter mainly in that they are not self-limited and do not grow by expansion, that is, by pushing aside the adjacent structures. The atrophy of the basilar portion of the cochlea, frequently associated with otosclerosis, is probably caused by the same (unknown) factor which causes the formation of the preotosclerotic changes.

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DIAGNOSTIC VALUE OF THE LATENCY PERIOD AFTER CALORIC STIMULATION OF VESTIBULAR APPARATUS

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THE "LATENCY period" is the time which elapses from the end of the thermic (caloric) stimulation of the ear up to the start of the tonic vestibular reflexes, as nystagmus, deviation reaction, and arm tonus reaction.[1] This period is, according to my experiences, of importance and help diagnostically. Other physicians share this view. On the other hand, Jongkees [2] doubts its value but measures it in every case with the stop watch. Arslan [3] denies the latency period any significance for the semeiology of the vestibular apparatus. He does so chiefly for the reason that the length of the latency period is not dependent on changes in the peripheral vestibular apparatus but rather on anatomic factors, as the extension of pneumatization of the osseus antrum bridge, and, in a less degree, on the speed of the blood stream, as proved by Schmaltz [4] and Dohlman.[5]

Arslan uses the method of Veits [6] for the syringing of the ear, starting in the indifferent position of the horizontal canal (the head of the test person bent forward at an angle of 30 degrees). When the syringing is finished, the head of the patient should be bent backward at an angle of 90 degrees, and the start of the nystagmus and so on observed. Nystagmus begins in normal persons after a very short latency period of not more than 1 to 2 seconds. According to Arslan,[7] only this latency period has some clinical value. He called it the "true latency period" or the "endolymphatic time." To this opinion of Arslan's I want to raise the following objections: 1. We all know patients with a strikingly shortened latency period and very strong vestibular reactions who rightly received the diagnosis of vestibular hyperexcitability. But some time later the same patients (syringed by the same method) show a normal latency period and also normal vestibular reactions. In other words, the former hyperexcitability changed to a normal response. Because the anatomic structure of the bony antrum bridge did not change in these patients

1. (*a*) Wodak, E.; Acta oto-laryng. orient. **2**:10, 1946; (*b*) Acta oto-laryng. **34**:593, 1946; (*c*) Pract. oto-rhino-laryng. **9**:30, 1947; (*d*) Vestibular Apparatus as Organ of Sense, Arch. Otolaryng. **46**:386 (Sept.) 1947.

2. Jongkees, L. B. W.: Which is Preferable Method of Performing Caloric Test? Arch. Otolaryng. **49**:594 (June) 1949.

3. Arslan, M.: Report for the Commission for the Standardization of the Vestibular Examination, Collegium Oto-Rhino-Laryngologicum, Rome, personal communication to the author.

4. Schmaltz, G.: Proc. Roy. Soc. Med. **25**:359, 1932.

5. Dohlman, G.: Acta oto-laryng. (Supp.) **5**:1, 1925.

6. Veits, C.: Zentralbl. Hals-, Nasen-u. Ohrenh. **17**:481, 1931.

7. Arslan, M.: Personal communication to the author.

in the time between the two examinations, we are, I dare say, entitled to ascribe the difference in the latency period to changed conditions in the peripheral vestibular apparatus itself (cupulae, maculae). 2. With Arslan's "true latency period" of 1 to 2 seconds' duration only, there is hardly any possibility of a striking shortening of this short span of time allowing, together with other symptoms, the diagnosis of hyperexcitability. On the other hand, we can observe that any lengthening of this "true latency period" enables us to diagnose—again together with other symptoms—hypoexcitability. Thus, the method of Veits-Arslan is certainly useful in the diagnosis of a peripheral hypoexcitability but, in my opinion, of little value in the diagnosis of a peripheral hyperexcitability.

For a better understanding of my subsequent remarks, it is advisable for me to report my technique of calorization and its results in the examination of the vestibular apparatus in normal test persons. Before the calorization, every patient should be examined to see if there are any spontaneous vestibular symptoms, as nystagmus, deviation reaction of the arms, or arm tonus reaction. If there are any, they must be registered. Then the test is started. The patient sits, his head in a normal upright position, wearing Bartels spectacles. The ears are syringed according to the method of Demetriades-Mayer with cold water (20 C.) or warm water (44 C.) by means of a syringe for 10 seconds, with the stream of water directed toward the posterior wall of the meatus externus. The syringing finished, a stop watch is started and the time of the appearance of the first slow component of the nystagmus is noted. The patient looks straight forward and examination is made for the appearance of the second-degree nystagmus.

The deviation reaction is examined in the following manner: The patient, sitting with closed eyes, extends both arms horizontally with the thumbs up; the physician, sitting opposite the patient, holds his hands exactly opposite to the patient's hands, controlling the direction and extension of the deviation of the arms. The arm tonus reaction can be examined with the test person in the same position with the palms down. Then all tonic vestibular reflexes—nystagmus, deviation reaction, and arm tonus reaction—are examined alternately until all phenomena disappear. The moment of disappearance should be determined by the stop watch separately for every reaction and registered. All this can be done by the physician alone.

After the cold-water syringing the latency period lasts about 15 to 20 seconds. Nystagmus, deviation reaction, and arm tonus reaction normally start at the same time and also last roughly an equal length of time, i. e., 60 to 90 seconds. Nystagmus and the other tonic reflexes reach their greatest strength about 30 seconds after their onset. At that moment the horizontal-rotatory nystagmus shows its jerks very distinctly. There exist, unfortunately, no reports about mass examinations of normal test persons stating the average values of the deviation of the arms to one side (deviation reaction) and difference between the height of the arms (arm tonus reaction). However, experience with many thousands of patients justifies calling a lateral asymmetric deviation of both arms of 10 to 20 cm. (4 to 8 in.) and a difference in the height of the arms (arm tonus reaction) of about 5 to 10 cm. (2 to 4 in.) normal. When warm water is used for syringing, the latency period is normally a little longer (20 to 25 seconds) and all tonic reflexes last a shorter time.

I want to stress how important it is to examine in every case besides the nystagmus the other tonic reflexes, as deviation reaction and arm tonus reaction. The deviation reaction of the arms is, in my opinion, besides another complicating psychological factor, the shifting of the subjective median plane of the test person, the chief reason for the past-pointing of the arms in the test of Bárány. This fact I hope I have proved in my monograph about the past-pointing test of Bárány.[8] In this monograph I suggested we abandon this test and replace it by the examination of the deviation reaction of the arms. This examination not only is very simple but also gives excellent and absolutely reliable results, demonstrable in 100% of normal persons provided, of course, the right technique is used. Also the arm tonus reaction should be examined in every case, although the percentage of normal persons with a clearly demonstrable arm tonus reaction is much less than 100%.

In the quarter of a century which elapsed since my recommendation was made, it has generally met with approval. The Commission for Standardization and Simplification of the Vestibular Examination (chosen by the Collegium Oto-Rhino-Laryngologicum) seems to have abandoned the past-pointing test of Bárány as a method of examination of the vestibular apparatus. Instead, the examination of the Deiterospinal reflexes, of which the deviation reaction is the most important, is recommended in the report of its chairman, Cambrelin, as an inseparable part of every normal vestibular examination.

DEVIATION FROM THE RULE REGARDING THE LATENCY PERIOD AND THE TONIC REFLEXES

Every striking shortness of the latency period (in repeated examinations) under 10 seconds or every lengthening of the latency period over 30 seconds must be considered as pathological. (Hyperexcitability must be divided into a peripheral form, caused by a change in the vestibular organ itself, and a form caused centrally. Central means from the entry of the vestibular nerve into the brain up to Deiters' nucleus and the central pathways.)

Peripheral hyperexcitability as a clinical picture is familiar to every otologist. We can make the diagnosis when the latency period is shortened to 10 seconds or less, the tonic reflexes (nystagmus, deviation reaction, and arm tonus reaction) being as a rule very strong. Sometimes these reflexes last longer than normal. There are also usually some vertigo and nausea.

For a better understanding of the following discussion on central hyperexcitability, a few preliminary remarks are necessary. All the reactions we observe in the vestibular examination of normal persons are by no means normal physiological reactions but must be considered as already pathological ones endangering the equilibrium of the test person. To protect this equilibrium, there are in different places of the brain (Bechterew's commissure, frontal lobe, cerebellum etc.), checking mechanisms which by their quick intervention restore the disturbed equilibrium, suppressing the reactions. This holds true not only for the objective vestibular phenomena (nystagmus, deviation reaction, and arm tonus reaction) but also for the subjective ones, as vertigo or sensation of rotation.

8. Wodak, E.: Der Bárányesche Zeigeversuch: Seine physiologischen Grundlagen und klinische Methodik, Vienna, Urban & Schwarzenberg, 1927.

I believe that every particular vestibular reaction has several such mechanisms of checking, one subordinated to the other. If one or several of these (really physiological) mechanisms are not functioning owing to an inflammation, a tumor, etc., then the reaction concerned will be free of restriction (*Enthemmt*). It will be exaggerated and last for a strikingly long time.

The checking mechanisms in the vestibular sphere are, in my opinion, of great physiological importance; were there none, our vestibular apparatus would be irritated by the most insignificant movements, as rotation or tilting of the head or body, in such a manner that we would permanently suffer from disturbances of the equilibrium and vertigo.[1d] Only the checking and suppressing of these small irritations make our life with its everyday movements possible.

Our findings in central hyperexcitability are: (*a*) a normal latency period (15 to 20 seconds) and (*b*) strikingly prolonged (three to five minutes) and at the same time exaggerated tonic reflexes. In other words, the peripheral vestibular apparatus answers to the caloric stimulation in a normal manner (normal latency period); whereas, owing to the elimination of one or several mechanisms of checking, the reflex concerned is free of restriction and lasts, therefore, extremely long. It is not necessary that in such cases all tonic reflexes should last simultaneously abnormally long. There may be a dissociation of them: the nystagmus may last strikingly long, while the deviation reaction and arm tonus reaction may be of normal duration. Also, the contrary behavior is possible. Such a dissociation of the tonic reflexes may sometimes give us hints for topical diagnosis.[9]

We must, of course, be very cautious in our judgment of a shortened or prolonged latency period as well as of a prolonged duration of the tonic reflexes. Only after repeated examinations and really striking deviations from the norm should these reactions be considered as pathological.

In this connection one may recall that, many years ago, G. Alexander and H. Brunner[10] also distinguished between a peripheral hyperexcitability (*periphere Übererregbarkeit*) and a central increased sensibility (*Überempfindlichkeit*). To distinguish both phenomena they used criteria similar to mine, of course without interpolation of the mechanisms of checking. All of us came to similar results, only in different ways. Similar mechanisms are found in neurology: exaggerated foot reflexes, culminating in the foot clonus, are, owing to the elimination or, at least, dysfunction of a central mechanism of checking.

To illustrate my views I want to record the following cases which, although there were no autopsies, nevertheless seem to support, even to confirm, to a certain degree my opinion.

CASE 1.—A middle-aged woman suddenly had violent attacks of vertigo. The vestibular examination showed a normal latency period but very prolonged nystagmus and deviation reaction (up to four minutes). Some time ago Halpern, of Jerusalem,[11] found that in women attacks of vertigo were caused by previous treatment with hot air for permanent waves at a hairdresser. Then I [11a] pointed out that in such cases the cause of the vertigo is by no means a peripheral affection of the vestibular apparatus but certainly a central nervous system damage in the brain (Deiters' nucleus, midbrain?) due to overheating. Fortunately, these damages are mostly reversible.

9. Wodak, E.: Acta oto-laryng. **9**:245, 1926.
10. Alexander, G., and Brunner, H.: Ztschr. Hals-, Nasen-u. Ohrenh. **3**:243, 1922.
11. Halpern, L.: Harefuah **22**:69, 1942.
11a. Wodak, E.: Harefuah **23**:57, 1942.

When I examined the above-mentioned woman, I remembered these findings, all the more because the vestibular findings pointed to central nervous system damage. I asked the woman if she had recently had a treatment with hot air at the hairdresser. She declared that about half a day before the onset of the vertigo she had such a treatment. She recovered completely in a few days, a further proof that there were probably no irreparable central nervous system damages (hemorrhages, edema, etc.).

The symptoms of the patients reported below resembled those of heat stroke; both patients suffered from vertigo, heaviness of the head, etc. I believe that probably the pathologicoanatomic findings would also be very similar, if not identical; and so would be, in my opinion, the vestibular findings. Until this time, I had no opportunity to examine the vestibular apparatus of a person with such a heatstroke.

Case 2.—A middle-aged man complained of attacks of vertigo for some months. The vestibular examination showed a normal latency period and extremely prolonged tonic reflexes (up to four minutes). Although there was no question of a treatment with hot air, I suspected, nevertheless, some connection with heat and asked if he perhaps dealt with heat in his profession. He said that he owned a steam laundry located in a small room emanating terrible heat. He had always suffered much from it in our subtropical climate. In my opinion, in this patient also the heat factor may have been chiefly responsible for the attacks of vertigo.

Case 3.—A man 20 years of age with purulent chronic otitis of the right ear suddenly became ill with vertigo and fever but without any pains in the right ear or mastoid. The ear itself showed no sign of exacerbation or symptom of fistula. There was also some hearing for whispered voice in the right ear, and the patient had on both forearms a rash which looked toxic. The vestibular examination showed a normal latency period but abnormally prolonged tonic reflexes (more than four minutes). Based on the latter findings, I conjectured that the patient had an irritation of the central vestibular apparatus caused by a toxic infection. The further course showed that the patient had indeed something like an infectious disease (virus?). On the administration of penicillin the symptoms, including vertigo, fever, rash, and headaches, disappeared in a few days. The findings in the right ear remained unchanged; therefore, the assumption of a labyrinthine complication improved by penicillin can be dropped. Had I not suspected a central vestibular irritation, based on the vestibular examination, certainly I should have suspected a complicating labyrinthitis.

Peripheral hypoexcitability is diagnosed if the latency period lasts much longer than normal—approximately 30 seconds or more—and the appearing tonic reflexes (nystagmus, deviation reaction, and arm tonus reaction) are very weak and shorter than normal. These findings are explained by the assumption that the peripheral vestibular organ is answering, for reasons hardly known to us, to the caloric stimulus to a much less degree than normal. Therefore, the tonic reflexes not only appear much later but also are much weaker and of a shorter duration than normal. The last fact, i. e., the early suppression of the tonic reflexes, is owing to the normal, physiological action of the mechanisms of checking.

Central hypoexcitability until now could hardly be diagnosed. We could only conjecture that it existed when other symptoms were present (tumor, central degeneration or inflammation, etc.). Not long ago, I had the opportunity to see a patient with very serious troubles with his equilibrium after treatment for three months with streptomycin. When I saw the patient for the first time, it was many months after termination of the streptomycin treatment, and his unsteadiness in walking had already partly improved owing to compensation.

Case 4.—A man aged 40 received streptomycin because of an abscess of the liver. The exact quantity of streptomycin received could not be ascertained. The vestibular findings were no

spontaneous signs from the vestibular apparatus, no nystagmus, deviation reaction, or arm tonus reaction. The hearing was satisfactory. Expecting an inexcitability of the vestibular apparatus, I made at once a mass irrigation with 50 cc. of cold water (instead of the 20 cc. normally used) in the left ear; after a 60 second latency period there was a distinct slow component of the nystagmus to the left, later followed several times by a very weak quick jerk. There was, on the other hand, a definite deviation reaction of both arms to the left after only 25 seconds latency period. This deviation reaction was also very weak. Both phenomena grew stronger after another 30 seconds, being, however, always by far weaker than normal. The nystagmus and deviation reaction lasted for a strikingly long time, the nystagmus four minutes and the deviation reaction five minutes. The examination of the right ear gave nearly the same result.

We all know very well cases of total or nearly total elimination of the function of the vestibular apparatus after prolonged treatment with streptomycin. The majority of the authors, such as Fowler, and Hulk and Jonkees,[12] explain this as a central nervous system damage, probably in the region of Deiters' nucleus; but there are also authors, as R. Caussé,[13] who assume a peripheral damage in the cupulae and maculae. In my opinion, the central nervous system damage genesis is more probable, and, assuming the latter, one may perhaps explain the strange behavior of the tonic reflexes in the following way: The central nervous system damage probably put out of action one or several of the mechanisms of checking. Therefore, the tonic reflexes—in this patient nystagmus and deviation reaction—were not immediately suppressed as they would be if the mechanisms of checking were intact. Notwithstanding their weakness, they lasted longer than in peripheral hypoexcitability.

Taking the above explanation for granted, we may perhaps also conjecture a central hypoexcitability in other patients with a similar behavior of the vestibular reactions.

86a Rotschild Blvd.

This manuscript was revised by Edgar I. Steinberg, M.D., M.Sc. (Med.), Philadelphia.

12. Hulk, J., and Jongkees, L. B. W., abstracted, Excerpta Med. (Sect. 11) **3**:271, 1950.
13. Caussé, R.: Ann. oto-laryng. **66**:518, 1949.

Case Reports

ABERRANT SALIVARY GLAND TISSUE IN A TONSIL FOSSA

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IT IS UNUSUAL to find salivary gland tissue lodged in the tonsil fossa after the tonsil itself has been removed. The color and texture are similar to those of the tonsil; however, the lobulated segments should soon make the surgeon suspicious. Neumann [1] in 1928 reported the first case of such tissue in the tonsillar fossa. In 1948 O'Neil [2] reviewed the literature and added another case of his own. He noted the cases of Neumann,[1] Tillotson,[3] Sydell,[4] Prohovnik,[5] Armstrong,[6] and Hitschler and Cope.[7] There were four cases mentioned by Warren B. Davis and Joseph C. Donelly, which were reported in the discussion following the report by Hitschler and Cope [7] in 1940. Another was reported by Peele [8] in 1951, making a total of 12 cases which have been reported in the past. The following case will be the 13th to be reported.

REPORT OF A CASE

R. L., a 57-year-old white woman, had been having pain and poor vision in the left eye for 23 days. Her ophthalmologist was treating the eye for acute hyalitis with cortisone drops. The patient was sent to an internist for a general check-up and to me to rule out a focal infection in her head. Her history revealed that when she was 18 months old she had placed a corkscrew in her mouth. This became lodged in her throat, and a forceful effort was required to remove it. She had had frequent pains in her left chest with shortness of breath for about six weeks. For years she had been aware of a small mass in the right thyroid lobe. She had had frequent migraine headaches since the age of 18. Examination of the ears, nose, and throat revealed the presence of cryptic, slightly enlarged tonsils. Caseous debris was expressed from the crypts on pressure. The patient was edentulous. The ears, nose, and sinuses were normal. A 2-cm. mass was palpated over the right lobe of the thyroid gland. A diagnosis of chronic tonsillitis and nontoxic nodular goiter was made, and tonsillectomy recommended.

The patient was hospitalized on May 16, 1951. Findings on routine laboratory studies were within normal limits. A local tonsillectomy was performed. After the left tonsil had been removed, a grayish-white lobulated tissue was seen extending approximately 2 cm. from the

1. Neumann, H.: Schleimdrüse von Tonsillenbette ausgehend, Wien. med. Wchnschr. **78**:650 (May 12) 1928.

2. O'Neil, J. J.: Aberrant Salivary Gland Tissue in Tonsillar Fossae: Review of Literature and Report of Case, Eye, Ear, Nose & Throat Month. **27**:234 (May) 1948.

3. Tillotson, R. S.: Aberrant Salivary Gland in Tonsil Fossa: Report of a Case, California & West. Med. **33**:744 (Oct.) 1930.

4. Seydell, E. M.: Parotid Gland Tissue in Tonsillar Fossa, Ann. Otol. Rhin. & Laryng. **43**:304 (March) 1934.

5. Prohovnik, J.: Salivary Gland Tissue in Tonsillar Fossa, Ann. Otol. Rhin. & Laryng. **45**:1181 (Dec.) 1936.

6. Armstrong, G. G.: Salivary Glands as Related to Otolaryngology, Mississippi Doctor **19**:493 (March) 1942.

7. Hitschler, W. J., and Cope, T. A.: Aberrant Salivary Gland in Tonsillar Fossa, Arch. Otolaryng. **34**:174-176 (July) 1941.

8. Peele, J. C.: Uncommon Conditions in the Field of Otolaryngology Seen in Office Practice, A. M. A. Arch. Otolaryng. **53**:492-514 (May) 1951.

center of the tonsil bed to the infratonsillar area. This tissue was grasped with the tenaculum and snared out. This procedure was repeated until a deep recess was made into the neck. As piece after piece was removed, it became apparent that the mass was larger than it had first appeared. The tissue extended into the region of the great vessels of the neck. When a deep segment of the tissue was snared, sudden bleeding began. After the bleeding area was sutured the operation was stopped. The tonsil and aberrant tissue were submitted for pathology study with a tentative diagnosis of parotid gland tissue.

Pathologist's Report.—The specimen consisted of a mass of tonsillar tissue approximating 2.0 cm. in extent, together with a mass of brownish tissue composed of numerous angular-shaped nodules characteristic of salivary gland tissue. This tissue mass measured 2.5 cm. in greatest dimension. This also was stated to be from the left tonsillar fossa. In a separate container was another mass of connective tissue and tonsil stated to be from the right side.

Microscopic Description: The tonsil tissue on each side exhibited some evidence of inflammatory involvement of chronic type associated with considerable fibrosis. The salivary gland tissue was poorly differentiated, resembling embryonic type of gland pattern. There was no evidence of neoplasm.

Microscopic Diagnosis: The diagnosis was (1) bilateral chronic tonsillitis with fibrosis, and (2) salivary gland elements.

Postoperative Follow-Up.—The tonsil fossae healed without incident. The patient has experienced no recurrence of the eye trouble. The Tumor Board reviewed the case and advised that further surgery was not indicated.

SUMMARY

A case of aberrant salivary gland tissue in the left tonsillar fossa is reported.

233 A St.

SALIVARY GLAND AND DUCT CALCULI

JOSEPH H. MELANT, M.D.
BUFFALO

SALIVARY calculi certainly are not uncommon. However, a stone measuring 3.5 cm. by 1.5 cm. by 1.0 cm. and weighing 4.0 gm. situated in Wharton's duct is rare. Just as the length of the removed stone was excessive, inversely, the length of the patient's history was brief. That contrast is the basis for this report.

REPORT OF A CASE

E. C., a 34-year-old well-developed man was referred to me by a dentist because of pain and swellings under the tongue and in the left submaxillary region. The patient was an intelligent young man, and there was no reason to doubt the following history. Ten days previously he had noticed a swelling under the tongue on the left side. About two days later, a tender mass developed near the angle of the lower left jaw. He could move the tip of his tongue upward and backward, but forward movements caused excruciating pain. After two penicillin injections (a total of 800,000 units) the patient was referred by the family physician to his dentist.

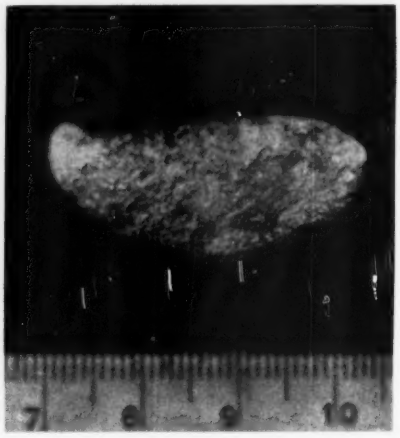
After a careful examination of the patient's oral cavity, including an occlusal roentgenogram of the floor of the mouth, the dentist referred the patient to me. Examination revealed a red, tender swelling in the floor of the left side of the mouth extending from the lateral incisor to the first molar. A corresponding swelling was present in the left submaxillary region. Gentle pressure upon this swelling caused frank pus to exude from the orifice of Wharton's duct at the frenum of the tongue.

With the area under local anesthesia an incision approximately 3 cm. long was made over the sublingual mass in the direction of the submaxillary duct. The stone was removed and the incision left open. No drain was inserted. Penicillin was administered intramuscularly and gantrisin® (3,4-dimethyl-5-sulfamido-izoxazole) and alkaline mouth wash were prescribed. Two days postoperatively, the sublingual edema had disappeared and the size of the submaxillary gland was greatly diminished. One week later, normal-appearing saliva could be expressed from the submaxillary duct that was operated on.

Salivary calculi are found in the following order of frequency: submaxillary duct, submaxillary gland, parotid gland, parotid duct, and sublingual gland. Stones are almost never found in the sublingual ducts because of their multiplicity and consequent ability to secrete saliva, even though the main duct (Bartholin) may become obstructed. Gland calculi are usually larger, although in this case the stone was found in the duct. Several factors may account for the higher incidence of concretions in the submaxillary gland and duct. Anatomically the orifice of the duct, although situated beneath the tongue, is nevertheless exposed to a considerable amount of trauma in the normal process of mastication. Tartar is most frequently formed about the necks of lower incisor teeth on their lingual surface. Small pieces of tartar could very well be forced into the duct, laying the nucleus of a subsequent calculus. The flow of saliva through Wharton's duct is considerably slower than that of saliva coming from the parotid gland for several reasons: (1) The submaxillary duct is wider and slightly longer than the parotid duct; (2) the gland itself is situated below its duct, and consequently the secretion must travel "up-hill" as it were; (3) physiologically the parotid secretion is much less viscid, since it contains no mucin.

Although Stensen's duct possesses a rather sharp bend at the edge of the masseter muscle, where it pierces a fat pad and the buccinator muscle to enter the oral cavity, this does not compare with the irregularities, diverticula, and constrictions (for example, the passage of the lingual nerve under the duct) encountered in the route of the submaxillary duct. These irregularities may serve as sites for a stasis and a subsequent growth of micro-organisms, with eventual stone formations.

There are several popular theories of stone formation, and almost all deal with a precipitation of calcium salts about some nucleus. Kuttner states that this nucleus is epithelial debris, while Soderlund has demonstrated an Actinomyces as the nucleus. Many cases of a foreign-body nucleus have been reported. Finally, Kirk has advanced the following theory: Escaping carbon dioxide from a solution in the mouth causes a precipitation of calcium salts in the presence of colloid mucin; eventually a calcific concretion forms. The chemical composition of salivary calculi is approximately as follows: calcium phosphate 65%, organic matter 25%, calcium carbonate 6%, and traces of iron and magnesium.



Stone removed from Wharton's duct.

Although the incidence of stone formations is greater in men than in women, the question of whether it occurs more frequently in smokers remains moot. Treatment depends largely upon the site and size of the calculus. Concretions in any of the ducts may be removed intraorally, and the larger the stone the easier its removal. If a small stone cannot be milked out of its duct, a suture should be placed behind the stone to prevent its sliding back before the duct is incised. Frequently it is possible to remove a gland stone intraorally. Multiple concretions and repeated infections necessitate an external operation with removal of the gland. In the case of the parotid gland, great care should be exercised to avoid injury to the facial nerve.

The classic history of periodic glandular swelling at mealtime is due to a ball-valve type of action. Persistent swelling points to chronic infection, which may destroy the alveolar structure of the gland. In extreme cases, fixation of the floor of the mouth with cellulitis of the neck may be found.

1105 Broadway (12).

Sister Mary Vincentia of the Mercy Hospital, Buffalo took the photographs.

Abstracts from Current Literature

Ear

ELECTROENCEPHALOGRAPHY IN THE DIAGNOSIS OF HEARING LOSS IN THE VERY YOUNG CHILD. R. E. MARCUS, E. L. GIBBS and F. A. GIBBS, Dis. Nerv. System **10**:170 (June) 1949.

Education of the hearing-handicapped child, to be effective, must be instituted early, but the diagnosis of profound deafness in the very young is clinically difficult. The authors report an approach utilizing the "arousal response" (waking activity interrupting cortical sleep patterns) in the electroencephalogram of the sleeping child. Various loud, frightening noises were produced, and it was observed that the "arousal response" was obtained when the sound was perceived and was the same as that caused by other types of sensory stimulation. Nineteen patients, from 17 months to 9 years of age, were studied; in 13 no audition could be demonstrated by routine otological testing. Four of the latter group showed some "arousal response." It is concluded that electroencephalography provides a technic for the diagnosis of deafness in the infant and child and that the "arousal response" to appropriate stimuli is an adequate index of hearing. As an additional observation, it was found that of the 13 children with recognized deafness, seven had electroencephalographic abnormality. The remaining six patients, in whom the primary disorder was retarded speech and an associated incomplete reduction in auditory function, all displayed electroencephalographic irregularities. The relation of hearing loss to disorders of the central nervous system should receive further investigation.

BEATON, Tucson, Ariz. [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

ROLE OF THE VESTIBULAR APPARATUS IN THE PRODUCTION OF NAUSEA AND VOMITING FOLLOWING THE ADMINISTRATION OF MORPHINE TO MAN. A. RUBIN and J. WINSTON, J. Clin. Invest. **29**:1261 (Oct.) 1950.

Morphine has among its disturbing side-effects the production of nausea and emesis. Studies have suggested that morphine may disturb vestibular function and that the vestibular activity incident to ambulation may then result in an increase in nausea and emesis. To investigate this possibility, the authors examined the responses to vestibular stimulation in 20 healthy adults before and after the administration of morphine.

It was found that morphine sulfate produced a significant decrease in vestibular response. Vestibular stimulation following the administration of morphine sulfate resulted in a notable increase in nausea and emesis. Amphetamine sulfate was of no apparent benefit in relieving this emesis. Dimenhydrinate, given either orally or rectally, appeared to prevent and relieve nausea, vomiting, and vertigo following morphine, dihydromorphinone (dilaudid®), and meperidine, and its use therapeutically and prophylactically with these drugs is suggested.

Neither dimenhydrinate nor amphetamine altered the vestibular changes produced by morphine. Vestibular changes following morphine resulted in no significant changes in blood pressure or pulse rate.

ALPERS, Philadelphia [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

TUBERCULOUS OTITIS AND MENINGITIS. D. I. ARBUSE and E. LEVENSON, J. Nerv. & Ment. Dis. **113**:165 (Feb.) 1951.

Of a series of 42 patients with tuberculous meningitis, two had associated tuberculous otitis media. One, a man aged 46, had chronic otologic disease of three years' duration, and one year prior to his admission the aural discharge was found to contain tubercle bacilli. He died of lymphocytic meningitis, from which tubercle bacilli were not cultured. The second patient, a girl aged 18 years, had had a draining ear for 10 years, from which tubercle bacilli were ultimately cultured. Lymphocytic meningitis developed, and pellicle and Levinson tests gave positive results. She showed improvement for one year while receiving streptomycin therapy.

BERLIN, Chicago [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

HEARING AID AMPLIFICATION FOR OPTIMUM SPEECH REPRODUCTION. BOYD V. SHEETS, and LEROY D. HEDGECOCK, J. Speech & Hearing Disorders **14**:373 (Dec.) 1949.

One hundred five persons, 41 females and 64 males, ranging in age from 15 to 78 years with a mean of 49.17 years who had worn hearing aids at least one year, were tested individually over a three-hour period by means of audiometric pure tone, free field pure tones, speech reception thresholds with the Harvard spondees, and speech intelligibility test. There seems little justification for the use of selective amplification in fitting most hard of hearing persons with electronic aids. Best results in speech comprehension were achieved with a commercial instrument when it was adjusted to give an essentially flat response or a moderate high pass emphasis.

PALMER, Wichita, Kan.

SOME PRINCIPLES IN THE DEVELOPMENT OF SERVICES FOR CHILDREN WITH HEARING IMPAIRMENT. ARTHUR J. LESSER, J. Speech & Hearing Disorders **15**:101 (June) 1950.

Only a few hearing conservation programs have thus far developed as medical-care programs of the state boards of health and education departments. Most such departments are carrying out hearing-testing programs. During the past generation the hearing of several million school children has been tested by audiometers. Knowledge needed to develop comprehensive programs of audiology is available, but its application is still limited to relatively few children in large cities. Individualized programs must be developed with auxiliary workers available for rehabilitation.

PALMER, Wichita, Kan.

HEARING AID SELECTION BY UNIVERSITY CLINICS. RAYMOND CARHART, J. Speech & Hearing Disorders **15**:105 (June) 1950.

Carhart believes that hearing clinics in hospitals and universities should be centers where problem cases can be brought for analysis, guidance, recommendation, and remedial training. Such a clinic should be a center for advanced study of auditory difficulties.

PALMER, Wichita, Kan.

RELATION OF STREPTOMYCIN AND DIHYDROSTREPTOMYCIN TO HEARING AND THE VESTIBULAR APPARATUS. ARAM GLORIG, J. Speech & Hearing Disorders **15**:124 (June) 1950.

Streptomycin and dihydrostreptomycin given in doses of more than 1 gm. per day produce irreversible changes in both divisions of the eighth nerve in the majority of cases. Some such changes are produced on less than 1 gm. daily. All patients receiving either of these drugs for more than two weeks should have vestibular and auditory studies before and after treatment.

PALMER, Wichita, Kan.

USE OF CLINICAL PSYCHOLOGICAL SCREENING TECHNIQUES BY AUDIOLOGISTS AND SPEECH PATHOLOGISTS. HELMER R. MYKLEBUST, J. Speech & Hearing Disorders **15**:129 (June) 1950.

Myklebust suggests that a speech clinic should utilize certain psychological testing programs. These cover mental competence, social maturity, personality, motor ability, educational achievment, hearing and vision. The author suggests preferential tests.

PALMER, Wichita, Kan

AUDITORY COMMUNICATION. HALLOWELL DAVIS, J. Speech & Hearing Disorders **16**:3 (Jan.) 1951.

An all-around attack on the problem of auditory communication, involving the disciplines and technics of physics, physiology, anatomy, and psychology, is in progress. Although these border areas have their own language and concepts, they can be coordinated and unified by attention to the common concept of language as a tool of information.

PALMER, Wichita, Kan.

A Clinical Auditory Training Program. Albert F. Johnson, and Bruce M. Siegenthaler, J. Speech & Hearing Disorders **16**:35 (March) 1951.

An outline of 20 lessons given to members of an auditory training program is presented for use in group work with persons who have hearing losses. Lessons concern the care and use of the hearing aid, auditory discrimination tests, anatomy and physiology of the ear, phonetics of speech, and similar items. Many changes in the behavior, attitudes, and skills of patients occur during the training program. Reports from former patients are indicative of the general effectiveness of this program.

Palmer, Wichita, Kan.

Closure and Revision Following the Fenestration Operation. H. Davis, T. E. Walsh, and E. Eldert, Laryngoscope **60**:1181 (Dec.) 1950.

Davis and colleagues discuss the fenestration operation with special regard to closure of the fenestra. In most cases the fenestra closes at about the fifth month postoperatively. In 73 cases, not one closed later then 12½ months after operation. In each of three patients under the age of 15 years the fenestra closed. It is advised therefore not to operate on patients so young. In patients over 15, closure occurred nearly equally at all ages. Revisions were largely unsuccessful, as were those operations (not included in the above statistics) in which the cartilage stopple was employed.

Hitschler, Philadelphia.

Therapeutic and Toxic Effects of Streptomycin in Otology. Luzius Ruedi, Laryngoscope **61**:613 (July) 1951.

Although streptomycin has been used successfully in treating Pseudomonas infections of the ear, such as otitis externa and media, its most important use is in treatment of tuberculous otitis media. Tuberculous otitis (which is secondary to pulmonary tuberculosis in almost all cases) often starts with a severe earache but without any pronounced disturbance in the general condition of the patient. A few days later, purulent discharge is present, and soon there is a rapidly growing perforation. In about half of the cases, tubercle bacilli can be demonstrated. Deafness begins at a very early stage and becomes rapidly worse. It is a combination of perceptive and conductive deafness, the former being caused by a tuberculous irritation of the labyrinth.

The treatment of the condition consists in not only parenteral administration of streptomycin or dihydrostreptomycin but also instillation locally of an aerosol solution (10% streptomycin in water). Although others have reported a fair percentage of cases in which the perforation of the drum membrane healed, Ruedi did not find that to be true in his series of cases. However, there was definite improvement in 94 of 101 cases. Ear secretions ceased within one to two weeks. In some cases there was a recurrence, but treatment was again successful. The hearing loss, however, which had already occurred, was not reversible, and hearing remained at the pretreatment level.

There has been much discussion of the toxic effects of streptomycin. These effects can be minimized or eliminated by regulation of the dosage. Not more than 24 mg. of the drug per kilogram of body weight per day should be given. Dihydrostreptomycin is even less toxic than streptomycin. The symptoms of toxicity appear mainly related to the vestibular apparatus, and seldom is there a hearing loss from this cause. Ruedi describes pathological and histological changes in experimental animals, and illustrations are shown.

The article concludes with a discussion of the use of streptomycin to prevent attacks of vertigo in Ménière's disease. Some good results were obtained. The treatment should be instituted, however, only in cases in which the disease is resistant to the customary remedies.

Hitschler, Philadelphia.

Rapid Antibiotic Sensitivity Determinations for Pseudomonas Aeruginosa Isolated from External Otitis. Roland B. Mitchell, Anna C. Arnold, and James E. Lett, Laryngoscope **61**:649 (July) 1951.

Many cases of external otitis are caused by Pseudomonas aeruginosa. Filter paper discs saturated with various antibiotics of different concentrations were placed on the surface of agar

plates inoculated with Ps. aeruginosa. It was determined by photographs that in six to eight hours there were zones of growth inhibition present about the discs to which the organism was sensitive and that these zones were sufficiently definite to justify a report at this time.

HITSCHLER, Philadelphia.

EXPERIENCES WITH PARENTERAL VITAMIN A THERAPY IN DEAFNESS AND TINNITUS. SHIRLEY HAROLD BARON, Laryngoscope **61**:530 (June) 1951.

Thirty-six deaf patients were treated with a certain preparation of vitamin A parenterally. Results led to the conclusion that there was no direct benefit to either the hearing or the tinnitus. These unfavorable results are compared with the apparently favorable ones obtained by some other workers. It is noted that Perlman's work on dogs contradicted the previous assumption that aural changes from vitamin A deficiency were reversible. HITSCHLER, Philadelphia.

UNILATERAL DEAFNESS AND PROGRESSIVE FACIAL PALSY DUE TO INTRAPETROUS NEUROFIBROMA. J. GRAFTON LOVE, Proc. Staff Meet., Mayo Clin. **25**:228 (April 26) 1950.

Love reports the case of a girl aged 16 with deafness in the right ear of eight years' duration and progressive paralysis of the right side of the face for two and one-half years. The roentgenologic picture was that of an intrapetrous epidermoid. At operation the tumor was found entirely within the petrous bone. It proved to be a neurofibroma, very fibrous in type. Presumably, the tumor had taken its origin from the eighth cranial nerve within the petrous bone, and it never at any time presented in the cerebellopontine angle, as tumors of the eighth nerve usually do.

The facial paralysis and loss of hearing were unaltered by operation. One month after the first operation a spinofacial anastomosis on the right side was carried out. Three months later there was no definite clinical evidence of reinnervation except for possible slight movement about the right corner of the mouth and below the nose. Fourteen months later there was pronounced improvement, with movement of all facial muscles, including the forehead.

ALPERS, Philadelphia [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

INFLUENCE OF HEREDITY ON THE PNEUMATIZATION OF THE TEMPORAL BONE. HUGH W. SCHWARTZ, J. Laryng. & Otol. **65**:317 (May) 1951.

There is much confusion about pneumatization of the temporal bone and as to whether there is more than one normal type. Wittmaack stated that at birth the tympanic cavity, attic, and antrum are filled with a myxomatous tissue covered with cuboidal epithelium; during the first year the subepithelial tissue is absorbed and the epithelium lines these cavities. This epithelium-covered myxomatous tissue grows into the marrow spaces of the temporal bone, where the same process occurs, forming the mastoid air cells. He held that interference with this process by entrance of amniotic fluid, meconium, or other substances into the middle ear or by infantile otitis media arrests the pneumatization and results in diploic or sclerotic mastoids.

Smith agreed with most of these views but felt that the constitution of the osseous tissue, rather than infection, arrested development. Schwartz felt that the inherited quality of the myxomatous tissue determined the type of mastoid. Many facts are presented to throw doubt on these theories. It has been stated that over 90% of newborn infants have otitis media neonatorum. Roentgenograms of children who have had otitis media in infancy often show pneumatized temporal bones. In patients with acute mastoiditis who had no surgical treatment, pneumatized mastoids have later developed. Chronic disease in a pneumatized bone produces a fibrotic rather then a sclerotic mastoid.

Schwartz feels that heredity is the main factor influencing the type of temporal bones that will develop and adequately supports this idea by finding the same type of pneumatization in 19 sets of 25 sets of identical twins and by large-scale roentgenographic studies of family groups, which show a strong tendency for members of the same family to have the same type of temporal bone pneumatization.

LEJEUNE and LEWIS, New Orleans.

SURGICAL TREATMENT OF MÉNIÈRE'S DISEASE. R. ARANA INIGUEZ, P. HOUNIE, and J. SAN JULIAN, An. oto-rino-laring. Uruguay **20**:105, 1950.

The authors present the case of a patient with severe Ménière's syndrome in his left ear, with complete deafness in this ear. He had received every form of medical treatment, with no appreciable improvement in his condition. Finally section of the eighth nerve was performed, with a complete cure for a period of two years.

The authors describe various surgical technics that can be employed: (*a*) opening of the semicircular canal followed by either alcohol injection (Mollison) or electrocoagulation (Day), or (*b*) opening of the endolymphatic sac (extradural) in the posterior fossa (Portmann) or by a transtegmental approach in which the external semicircular canal is destroyed by electrocoagulation.

The neurosurgical procedure is either partial or total section of the eighth nerve. The indications for this are a suspected lesion at the cerebellar pontine angle, (2) abolition of vestibular function, and (3) presence of useful hearing. In such cases partial section of the nerve is indicated. However, where there is a profound deafness associated with vertigo, total section of the nerve is advocated.

PERSKY, Philadelphia.

SOME RECENT WORK ON AUDITORY ADAPTATION AND ITS RELATIONSHIP TO THE LOUDNESS RECRUITMENT PHENOMENON. C. S. HALLPIKE and J. D. HOOD, J. Acoust. Soc. America **23**:270 (May) 1951.

It is regrettable that so much of the literature on acoustics is over the head of the average reader, since the worth and the practical application of the information is missed. Authors should be particularly conscious of the fact that odd, singular, and peculiar words make for both annoyance and lack of clarity to the reader. Terms such as "per-stimulatory" and "on-effect normalcy" are somewhat difficult until one is thoroughly familiar with what they label.

The present article is a follow-up of valuable study on the loudness-balance phenomenon in Ménière's disease (a subject complex enough by itself), in which the conclusion was reached that the phenomenon was explained by disordered function of the hair-cells caused by the disease. Now the study shifts to another experimental approach to the problem of recruitment (loudness-balance), that of the function of "auditory adaptation." Two frequency oscillators (audiometers) were used, one for each ear. Duration of stimulus impulses were varied in one ear while continuous in the other. The fall in the balancing level indicated abnormal loss of sensitivity of the impaired ear.

Otologists interested in and truly familiar with the physiology of hearing will appreciate the ingenuity of the authors and the originality of their findings in the "lowering of the restoration rate of energy dissipated by activity."

MACFARLAN, Philadelphia.

USE OF PERFORATED ACRYLIC STOPPLE IN FENESTRATION OPERATION. WALDEMIR SALEM, Rev. brasil. oto-rino-laring. **19**:1 (Jan.-Feb.) 1951.

The author has devised a perforated acrylic mold from 1.5 to 2 mm. in diameter and of oval shape which he used in an attempt to prevent osteogenesis following a fenestration operation. The stopple is well tolerated by the labyrinth, and the sound waves seem to penetrate satisfactorily through the perforation. In one case in which this was used the audiometric curve showed that the patient maintained a very satisfactory improvement in hearing acuity six months after operation. The author states that, though he has not had sufficient cases to indicate the actual value of this modification, he believes that the results are very encouraging.

PERSKY, Philadelphia.

SOME CLINICAL OBSERVATIONS ON OTOGENIC DIGESTIVE TROUBLES. P. ROHMER, E. SCHNEEGANS, and C. WILD, Ann. pædiat. **175**:308 (Oct.) 1950.

Based on a few typical examples, this article suggests the relation between otitis and mastoiditis on the one hand and acute gastrointestinal disturbances on the other. The clinical picture is discussed, and conclusions are drawn with regard to treatment.

The authors excluded cases with acute toxicosis because in these other therapeutic measures had to be taken, thus increasing the difficulties in judging the results. But even with them the authors believe that some patients can be saved by timely antrotomy.

FROM THE AUTHORS' SUMMARY.

Pharynx

FAILURE OF TONSILLECTOMY. T. L. HYDE, J. A. M. A. **146**:1478 (Aug. 18) 1951.

Hyde observed 1,000 cases consecutively and without selection in a general surgical practice over a period of years. Detailed histories were recorded, including age of patient, age when tonsillectomy was performed, where the operation was performed, whether the surgeon was a "nose and throat" specialist, why the operation was performed, the anesthetic used, any benefit obtained, and whether the patient knew of any tonsillar tissue left. Then an examination of the pharynx was undertaken, and any tags present were recorded as grade 1 to 4, grade 1 being the smallest and grade 4 being an amount equal to the size of a normal tonsil.

The author deplores the failure to remove all the tonsil. He believes that the percentage of patients retaining tags is about equal whether the operation was performed by a general surgeon, general practitioner, the occasional operator or a specialist.

Here is his surgical technic for removal of the entire tonsil: "One begins the dissection with a sharp knife by outlining the tonsil, including all visible tonsil tissue, especially at the junction of the tonsil and tongue. The tonsil edge is grasped with a tenaculum at the lowest part, which anatomically is designated the superior pole. The dissection proceeds upward and backward by sharp snips with scissors. The line of dissection is always clear, because the blood runs downward and away from it. Spurting arterial bleeding may occasionally jet upward to obscure the line of dissection, and such bleeding is controlled by clamp and the tie as in any other operation. The clamp can be applied and left dangling until the dissection is complete. The Sten clamp with the jaws curved at 45 degrees seems most convenient. The tie is made with a 20 in. strand of No. 20 crochet cotton thread. No assistant is necessary. The dissection by scissors is carried completely through to the base of the tongue. No snare is used. There is no reason for one end of the tonsil being dissected any differently from the other end. . . ."

The technic described above is used for general tonsillectomy. For local tonsillectomy, Hyde uses virtually the same surgical technic except that the position of the patient is different and the operator sits in front of him and looks upward into his mouth.

Hyde states that in 99 per cent of the 262 cases reported he has been successful in performing a tonsillectomy that is free of tags or stumps.

GORDON, Philadelphia.

BROMIDE-IODIDE THERAPY IN DISEASES OF THE TONSILS OF CHILDREN. L. LANDUCCI, Clin. Pediat. **33**:117 (March) 1951.

Children with diseased tonsils have been treated by hydrotherapy at a bathhouse with mineral water containing bromides and iodides. Most of the 600 thus treated showed improvement after a period of 15 days. A tonsillar test is described: The white blood cell count is made before and 60 minutes after each tonsil has received a three-minute digital massage. The reaction is regarded as positive, and indicative of infection, if the white cell count has shown an increase or decrease of more than 1,000. Of 30 children tested, all gave a positive reaction before the hydrotherapy was begun. Only eight of them gave a positive reaction at the end of the course of hydrotherapy. The author ascribes the benefit of the treatments to a general constitutional reaction rather than to local effect on the tonsils.

HIGGINS, Boston.

HISTOPATHOLOGICAL STUDY OF TONSILS IN CHILDREN. T. PORCELLI, Minerva pediat. **2**:593 (Dec.) 1950.

From a careful study of the tonsils of 42 children the author has concluded that it is impossible anatomically to distinguish between simple hyperplasia and hyperplasia due to previous inflammation. He found no histological picture characteristic of any specific disease.

HIGGINS, Boston.

Larynx

TRACHEOTOMY FOR PREVENTION OF PULMONARY COMPLICATIONS IN POSTOPERATIVE AND SEVERELY DEBILITATED PATIENTS. JOSEPH P. ATKINS, J. A. M. A. **146**:241 (May 19) 1951.

Atkins emphasizes the importance of tracheotomy in cases in which obstruction in the upper or lower airway may threaten life by asphyxiation or by atelectasis and its sequelae. He proves his points adequately with citation of case histories.

The prevention of anoxic episodes, whenever their occurrence can be reasonably predicted, especially in extensive operations about the mouth, pharynx, or neck, may be obtained by an orderly tracheotomy.

The author emphasizes that tracheotomy is not a panacea, but in the majority of the cases in which it is indicated it offers a simple and positive means of protecting the respiratory tract and lessening the exhaustion that many severely debilitated patients experience—exhaustion which may turn the balance toward recovery or toward death. GORDON, Philadelphia.

EARLY DETECTION OF BRONCHIOGENIC CARCINOMA. D. L. PAULSON, and R. R. SHAW, J. A. M. A. **146**:525 (June 9) 1951.

The authors emphasize that the over-all salvage rate for bronchiogenic carcinoma remains disappointingly low despite advances in the surgical treatment. In their observation of 362 patients seen during the preceding three and one-half years, pulmonary resection could be applied to only 107 (30%). They make a plea for earlier detection, and they review briefly the symptomatology and the roentgenographic examination, illustrating how errors in diagnosis have taken place. In many such cases diagnosis has been made six months too late. Even the roentgen examination in the early stages of this disease may be misleading, and the clinical pattern is such that "the physician is frequently fooled early in the course of the disease and makes an erroneous diagnosis of asthma, bronchitis, virus pneumonia, unresolved pneumonia, tuberculosis or a fungus disease." GORDON, Philadelphia.

VOICE BREAKS AND PATHOLOGICAL LARYNX CONDITIONS. E. THAYER CURRY, J. Speech & Hearing Disorders **14**:356 (Dec.) 1949.

Voice breaks occur suddenly as breaks within a particular wave rather than as gradual changes occurring in inflectional variations in language. When subjects whose voices are "breaking" during puberty are compared with subjects in the literature who have organic anomalies, certain differences appear. The voices of normal subjects break downward nearly an octave, to a pitch approximately one-half that of the original level, while those of persons with organic defects tend to drop one-third of the original fundamental. Pathological subjects show considerable variation of the fundamental both before and after the break; they seem to evidence a typical breaking frequency for each individual. In contrast, normal subjects display a more closely knit vocal behavior. PALMER, Wichita, Kan.

Nose

RHINOPLASTY AND INTRANASAL SURGERY. L. W. EISENTODT, Eye, Ear, Nose and Throat Monthly **30**:413 (Aug.) 1951.

The author considers that the relation between rhinoplasty and nasal disorders is a more or less neglected question. His idea is that rhinoplasty should not be regarded as a purely cosmetic procedure, since many intranasal diseases and deformities require correction. He stresses the commonly held opinion that surgery on the nose should not be done during acute nasal infection, while operation is permissible in the presence of chronic or low grade processes. The common intranasal disorders amenable to surgery are outlined as atrophic rhinitis, chronic hypertrophic rhinitis, nasal tumors (as polyps, or hypertrophy of the nasal mucous membrane), nasal synechias, epistaxis, sinusitis, and hay fever or nasal allergies. In the latter conditions, it is the

author's custom to perform a rhinoplasty with submucous resection and complete resection of the anterior margins of both inferior turbinates. The desirability of combining intranasal and extranasal operations, rather than doing them separately, is stressed.

SELTZER, Philadelphia.

TREPHINE IN ACUTE FRONTAL SINUSITIS. D. MYERS, AND M. S. ERNSER, J. Internat. Coll. Surgeons **15**:686 (June) 1951.

The authors report 10 cases of frontal sinus disease treated by trephinement of the floor of the sinus, a method which has given them the best results. Antibiotic therapy precedes surgery, after which localization of the infection takes place, so that drainage is effected through the relatively avascular floor.

The technic advocated in this report aims to prevent the intracranial complications that sometimes occur, though this type of approach is not meant to supplant other surgical procedures.

GORDON, Philadelphia.

RHINOPLASTIC PROBLEMS CONFRONTING THE RHINOLOGIST. M. S. ERSNER, M. H. ALEXANDER AND D. MYERS, J. Internat. Coll. Surgeons **16**:165 (Aug.) 1951.

The authors discuss in moderate detail some of the finer points of nasal physiology, with emphasis on the numerous anatomic abnormalities encountered. They attempt to classify septal surgery into three eras, and they give a brief résumé of the rhinologists who pioneered certain techniques.

A general review of the anatomic relations is given with advice on managing reconstructive procedures along physiological principles and repairing injuries. They are helpful comments about correcting flaccidity when a complete submucous resection has been performed, and there is a discussion on the proper handling of other nasal deformities. A brief discussion follows concerning the physiologic function of the upper and lower lateral cartilages, their relation to the septum, and the numerous "little" procedures in the correction, positioning, and replacing of parts and segments to secure anatomic and physiologic integrity.

Advice is given on drooping of the nasal tip, septal perforation, air eddies, scar formation, contraction and synechia, scabs and crustations, and epistaxis.

It is rather difficult to abstract an article of this nature, since it deals with the manifold problems that one encounters in reconstruction of the internal and external nose and includes helpful hints and advice for remedying these problems. For a fuller appreciation, the reader is urged to study the original article.

GORDON, Philadelphia.

OSTEOMATA OF THE FRONTAL SINUS WITH SPECIAL CONSIDERATION OF THE SURGICAL REMOVAL. BENTON N. CLOVER, Laryngoscope **51**:341 (April) 1951.

The general subject of osteoma of the frontal sinus is reviewed. Various theories of pathogenesis, of which there are many, are mentioned, together with the histology and the classification. There may be just two kinds: one, the pedunculated hard tumor, and the other, the sessile soft growth. When the hard tumor is removed, underlying hard bone is reached, but after removal of the soft growth there is no underlying normal bone and one eventually arrives within the cranial cavity. The base of origin is discussed. The frequencies of these tumors is uncertain. Some men have seen many, while the author saw but two cases in his first 29 years of practice.

The treatment is surgical removal. However, if the tumor is small and is not pressing upon any vital structures, immediate removal is not necessary. X-ray examinations every two or three years may be resorted to. However, if there is any danger of the growth impinging upon vital structures or of secondary infection taking place because of limitation of sinus drainage, surgery should be done. A description of surgical procedures and techniques is included, together with six cases, each showing a clear-cut difference in its pathological, clinical and surgical details. In each of the cases the osteoma was of the pedunculated variety.

HITSCHLER, Philadelphia.

ELECTIVE RHINOPLASTY. H. O. BAMES, Plast. & Reconstruct. Surg. **8**:113 (Aug.) 1951.

The author has written an excellent manuscript on elective rhinoplasty, presented with 19 illustrations, 18 of which are in two-tone. Bames evaluated noses according to esthetic aberrations. Particular emphasis was placed on esthetic consideration. A careful medical history is necessary, and a physical examination is made to satisfy the surgeon that the indications for the operation are proper. The technical descriptions, including that of the actual operation and the profuse illustrations will reward a reader interested in this field of surgery. A description of the care of the wide alar base and wide nostrils, including comments on bleeding after a nose operation, is included. Postoperative care is also discussed.

SELTZER, Philadelphia.

THE RÉTHI INCISION IN RHINOPLASTY. HANS MAY, Plast. & Reconstruct. Surg. **8**:123 (Aug.) 1951.

The classic intranasal incisions after Joseph give in the majority of cases adequate access to the nasal framework for the various steps of rhinoplasty. In marked deformity of the lower or cartilaginous part of the nose, however, the Réthi incision has proved valuable. The author prefers a combination of the two and, with the use of eight illustrations and four case histories, proceeds to describe this technic.

The Réthi incision starts with a transverse cut through the columella. Before the incision is made, however, it is good to make a longitudinal scratch with the scalpel through the middle of the columella. This will facilitate proper alignment of the wound edges later. With the aid of traction sutures and sharp and, later, blunt dissection, the skin overlying the median crura and the lower lateral cartilages is mobilized and retracted until the upper lateral cartilages are reached. This exposure offers excellent access to and view of the entire lower nasal framework. This type of operation gives wide exposure for the correction of any of the components of the nose. A submucous resection of the septum may be added. The skin can now be draped back and sutured in place.

SELTZER, Philadelphia.

NEW INSTRUMENTS IN RHINOPLASTY. H. KOECHLIN, Plast. & Reconstruct. Surg. **8**:132 (Aug.) 1951.

Koechlin describes an instrument that he has utilized for narrowing the nose. This instrument enables one to achieve greater precision and thus to produce a fracture of the nasal bone exactly at the desired height. The author reports preoperative and postoperative observations in a series of cases in which this method was used. The instrument itself is described to consist of a handpiece, a drill, and a No. 14 gauge needle that is used as a guard for the drill.

SELTZER, Philadelphia.

RHINOMETRIC EXAMINATIONS OF SCHOOL CHILDREN WITH ADENOID VEGETATIONS. POUL STOKSTED, Acta otolaryng. **39**:44 (Feb.) 1951.

Nasal insufficiency of functional airflow can be determined by methods of rhinometry. This supplements objective examination. It can be used as a general screening test for school children to weed out those who require special examinations. The idea is not new, dating to the work of Brown and Claesenn in 1877. The indirect method of Zwaardemaker has been used a good deal since 1889. This method involves observing and measuring the patches of condensation on a plain mirror held just beneath the nares. All direct methods are based on a determination of the amount of air passing through the nose, the pressure in the nose during respiration, and the velocity of flow. Knowledge of any two of these factors makes the third a mathematical determinable. Respiratory curves can be kymographically recorded, thus establishing as well the needed time factor in calculations. In the author's method a fluid manometer records the ebb and flow of air, and this is transmitted to the sensitized film of an electrocardiograph. Respiratory curves are then calculated, starting from the basic formula V equals $H \times T \times D$, where V is respired volume, H is velocity of flow per second, and D is cavity lumen. T would equal time.

Results of the work have indicated that children with adenoidal obstruction have an insufficient airway; that the degree of this insufficiency is such as to cause an abnormal development in some cases of the mouth, throat, palate and chest contour. Testing with and without the use of ephedrine and the testing of normals all tend to confirm the accuracy of the method.

ROBERT LEWY, Chicago.

Miscellaneous

RIGHT AORTIC ARCH WITH A VASCULAR RING CONSTRICTING ESOPHAGUS AND TRACHEA, BÉLA HALPERT, WILLIAM T. SNODDY, KENNETH E. BOHAN, and CHARLES L. FREEDE, Arch. Path. **47**:429 (May) 1949.

Autopsy findings of a right aortic arch with a vascular ring are reported in two infant girls. For the first patient, 7 months old, the diagnosis of congenital heart disease was made at the age of 7 weeks, because of the findings of an enlarged cardiac shadow and right ventricular hypertrophy. After this there were repeated episodes of respiratory difficulties associated with spells of cyanosis. Two days before her last admission she was noted to choke with feedings. The second patient, 5 months old, was noted at the age of 10 weeks to have wheezing resiprations and a nonproductive cough. Feeding of 2 oz. (60 ml.) of formula caused regurgitation and at times initiated attacks of crying, cyanosis, and perspiration. Sleeping in an upright position seemed to alleviate difficulties. In the first case the diagnosis of a vascular ring constricting the esophagus was not made until autopsy. In the second case the clinical diagnosis was made at the last admission, and an attempt to relieve surgically the tension on the trachea and esophagus was successful. However, the patient's general condition was very poor, and she died eight hours after the operation. In the two cases findings were similar as regards the anomaly of the aorta. The ascending portion of the aorta arched over the right main bronchus and passed down to the right of the esophagus. From the arch arose, in order, left common carotid, right common carotid, and right subclavian arteries. The left subclavian artery arose in both cases from a medial diverticulum of the aorta (remains of the fourth arch). The left subclavian then continued into an obliterated ductus arteriosus, which connected with the left branch of the pulmonary artery. The arterial ring thus formed encircled the esophagus and trachea.

HAW CHAN, San Francisco.

PREVENTION AND TREATMENT OF MOTION SICKNESS. L. E. GAY and PAUL E. CARLINER, Bull. Johns Hopkins Hosp. **84**:470 (May) 1949.

Gay and Carliner report the results of their studies on the administration of dimenhydrinate (dramamine®).

During a 10-day sea voyage, dimenhydrinate was administered to 389 persons who suffered from seasickness. Of this number, 372 were completely relieved of symptoms within one hour after the first dose, of 100 mg. Seventeen persons derived only partial benefit or no relief. It was found that a dose of 100 mg. prescribed to be taken every five hours and before retiring was adequate to control the most distressing symptoms. When the patient was unable to retain a capsule, rectal administration proved effective. The benefit derived by this route was as rapid and as complete as that derived by the oral route. No untoward reactions to dimenhydrinate were encountered by any soldier who received the drug.

The action of dimenhydrinate on the prevention and control of motion sickness is not yet definitely established. Studies are in progress to attempt to explain the mechanism.

GUTTMAN, Wilkes-Barre, Pa. [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

ACTH AND CORTISONE IN ALLERGIC MANIFESTATIONS. S. M. FEINBERG, T. B. DANNENBERG, and S. MALKIEL, J. Allergy **22**:195 (May) 1951.

Observations on 15 patients who have severe asthma are summarized by the authors as follows: Corticotropin (ACTH) and cortisone are of appreciable value in the temporary treatment of certain types and phases of allergic syndromes. They are most helpful in intractable

asthma, in acute status asthmaticus, and in severe drug reactions. These hormones have a number of limitations in their usefulness. The length of remissions after a single course of treatment varies from a few days to a few months; the necessity for frequent injections is a handicap, and some types of allergy do not respond. Corticotropin and cortisone are not substitutes for more basic allergic management.

The dangers and hazards from continuous, and sometimes from temporary, use of these drugs are numerous, and the hormones must not be employed without a full understanding of their many actions. In patients treated with corticotropin or cortisone, the skin reactions to antigens and histamine remained unchanged. Quantitative studies of the ophthalmic and nasal reactions in those who have nasal and conjunctival allergy show the reactions not to be significantly altered. Sensitizing antibodies have remained unaffected after corticotropin or cortisone therapy.

FRIEDBERG, Chicago.

NASOPHARYNGEAL IRRADIATION. L. H. GARLAND, H. A. HILL, M. E. MOTTRAM, and M. A. SISSON, J. A. M. A. **146**:454 (June 2) 1951.

The authors discuss the relative advantages and disadvantages of x-ray and radium therapy in the treatment of lymphoid hyperplasia of the nasopharynx that causes deafness and allied auditory disturbances, illustrate the distribution of ionization through the involved tissues, and report some recent data on the dosage of beta and gamma rays from standard applicators. They feel that the dosage throughout the zone commonly involved in lymphoid hyperplasia is more homogeneous and efficient with external roentgen beams than with intranasal radium applicators.

In the treatment of 720 patients, excellent results were obtained with x-ray therapy in from 60 to 90%, depending upon the severity of the disease; these results were substantiated by the referring physicians and by repeated audiometric and other tests.

The maximum tissue dose required for cure when x-ray therapy is employed is much lower than when standard radium applicators are used. X-ray therapy is safer and should be the method of choice.

GORDON, Philadelphia.

PROGNOSIS OF SEPTIC THROMBOPHLEBITIS OF THE CAVERNOUS SINUS. H. I. LILLIE, J. Internat. Coll. Surgeons **15**:754 (June) 1951.

Lillie presents a lucid review of this important subject. History, anatomic background, symptoms and signs, and illustrative cases observed at the Mayo Clinic are cited.

The consensus at the present time favors the abandonment of surgical intervention in the treatment of fulminating thrombophlebitis, though for any localized infection drainage should be instituted. Appropriate chemotherapeutic agents, antibiotics, and anticoagulants should be administered at the earliest possible moment even though the diagnosis is not established.

It is of the utmost importance that these agents should be accurately administered under the guidance and supervision of persons skilled in their use. It is well known that chemotherapeutic drugs have been used indiscriminately for minor, self-limiting infections and that hypersensitiveness may sometimes be manifested.

Newer chemotherapeutic drugs and antibiotics may appear which will be even more effective than the ones presently in vogue.

GORDON, Philadelphia.

TECHNIQUES OF THERAPY FOR PRE-SCHOOL CHILDREN. JANE BEASLEY, J. Speech & Hearing Disorders **14**:307 (Dec.) 1949.

A combination of group and individual instruction two hours a day for a period of two weeks was carried out for four such two-week periods, a total of 16 preschool children being used. The results were the following: 1. Direct study of a child's behavior in interpersonal relations with his peers provides more adequate information on which to base procedures. (2) Direct clinical training can establish a broad base of experience-participation in group activity. (3) Direct clinical training for children enables parents to gain better understanding of their children.

PALMER, Wichita, Kan.

DIAGNOSIS AND TREATMENT OF A CASE OF HYSTERICAL APHONIA IN A THIRTEEN-YEAR-OLD GIRL. J. L. BANGS, and A. FREIDINGER, J. Speech & Hearing Disorders **14**:312 (Dec.) 1949.

A 13-year-old girl, with an I.Q of 87, had functional aphonia for seven years. The pattern of life of the parents was in general toward neurotic deviation. Hypnosis of the patient was unsuccessful. Her laryngeal musculature was flaccid. Ten and one-half weeks of therapy was sufficient to give propositional speech in all situations, and the patient has remained with normal vocalization for two years.

PALMER, Wichita, Kan.

LINGUAL ANOMALIES ASSOCIATED WITH PALATAL CLEFTS. MILDRED FREGURG BERRY, J. Speech & Hearing Disorders **14**:359 (Dec.) 1949.

Lingual anomalies frequently exist in cases of cleft palate. For a successful prediction of clinical procedures, an analysis of tongue function must be made.

PALMER, Wichita, Kan.

THE PITCH AND DURATION CHARACTERISTICS OF SUPERIOR FEMALE SPEAKERS DURING ORAL READING. JOHN C. SNIDECOR, J. Speech & Hearing Disorders **16**:44 (March) 1951.

Six carefully chosen superior women speakers read a 55-word prose passage under controlled experimental conditions. The data were compared with those previously reported for superior male speakers on the same material. Women's voices were pitched approximately two-thirds of an octave above men's voices. Voices of women were less variable in pitch than those of men. The mean extents of phonations, inflections, and shifts were found to be slightly less for women than for men, upward shifts exceeding downward shifts. Women's voices were found to have a less rapid mean rate of pitch change and notably shorter mean durations of phonation. Both male and female subjects used pitches below their singing range while talking.

PALMER, Wichita, Kan.

ARTIFICIAL STUTTER. BERNARD S. LEE, J. Speech & Hearing Disorders **16**:53 (March) 1951.

When a recording instrument is arranged so that speech is played back to the subject's ear at various time delays and the subject cannot hear his own voice at the time of speech, two reactions take place: Either the subject slows down and raises his intensity, or in attempting to maintain normal speed he halts or repeats syllables and continuous sounds. The delayed speech feedback provides vital information to speech theory, and the equipment provides the normal-hearing person with the experience of a problem resembling stuttering.

PALMER, Wichita, Kan.

Book Reviews

The New Way to Better Hearing. By Victor L. Browd, M.D. Price, $3.00. Pp. 226, illustrated, with 13 colored plates. Crown Publishers, Inc., 419 Fourth Ave., New York 16, 1951.

This book is written essentially for the hard-of-hearing person and devotes itself exclusively to what is generally called auditory training or hearing reeducation. It describes in detail, giving word lists and careful directions, how to make efficient use of residual hearing. Although the results claimed are somewhat too optimistic, this book fills an important void in the rehabilitation of the hard of hearing. It makes available to the public a concrete, easy-to-follow plan for hearing reeducation and brightens the outlook for many patients who do not have the benefit of training in large hearing centers.

In spite of overenthusiasm and exaggerated claims, this book is an excellent contribution to the field of otology.

Congreso panamericano de oto-rino-laringologia y broncoesofagologia. Price, not given. Vol. 1, Pp. 323; Vol. 2, Pp. 448. Garcia Morales-Mercant, S. A., Montevideo, Uruguay, 1950.

These two volumes of carefully assembled, well-printed articles presented at the Pan-American Congress in 1950 in Montevideo give an immediate appreciation of the surprising amount and the excellent quality of the work being done by our Latin-American neighbors. The material is too voluminous for a short review, but the evidence is apparent that the Congress is a strong and progressive organization.

The papers cover all phases of the three branches of otolaryngology, and most of the work is of the nature of original investigation of superior quality. There is a remarkable independence of thought and study given the subjects, and no accusation can be made that the authors are merely following the lead of the older groups in Europe and America.

The text is for the most part in Spanish, with an occasional English translation.

Audiometrie vocale. By J. E. Fournier, and S. A. Maloine. Price, 1800 francs. Pp. 93. Librairie Maloine, S. A., 27, Rue de l'Ecole de Medicine, Paris, 1951.

This book of 93 pages, is written by the clinical professor of otolaryngology of the Faculté de Médicine, Paris. It is a compendium of the literature on speech-hearing, practically all of which has originated in the United States over the last decade. It is clearly written, well illustrated, and thorough, yet it misses the recording of several of the advances made in this country along the lines of rating disability from deafness and auricular training.

The reason for the value of this volume is well expressed in the preface by Professor A. Auban of the same Faculté: "There is not a frank parallelism between frequency audiometry and testing with speech. Indeed, we have often examined a person with a mediocre audiogram and have been disturbed to find him hearing speech well. The reverse is also found."

Fournier has adapted the Harvard word list to the French language and covers interpretation testing in a practical way for French otologists. The recruitment phenomenon, dampening, and social adequacy testing are not touched upon, and there is a singular omission of the work on speech hearing done by Fowler, Sr., Bunch, Macfarlan, and other early American authorities. It is perhaps unavoidable, yet somewhat regrettable, that we find still another volume on hearing and acoustics written as if by an acoustic engineer, full of graphs and mathematical formulae which are practically incomprehensible to the average otologist.

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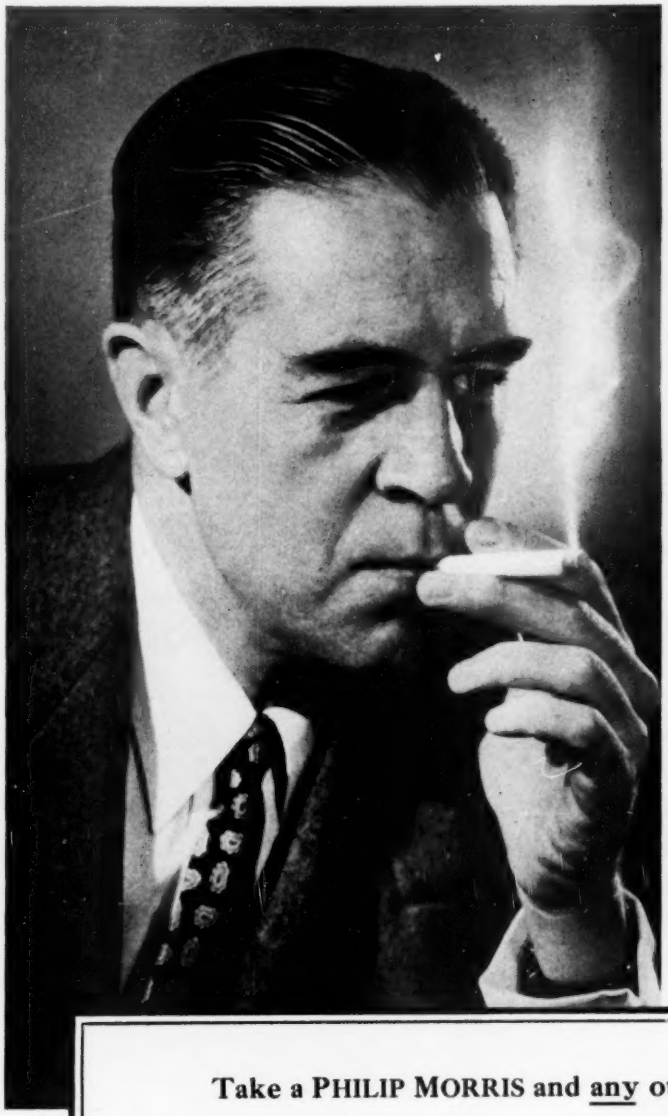

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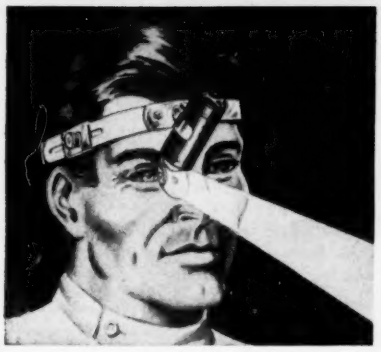

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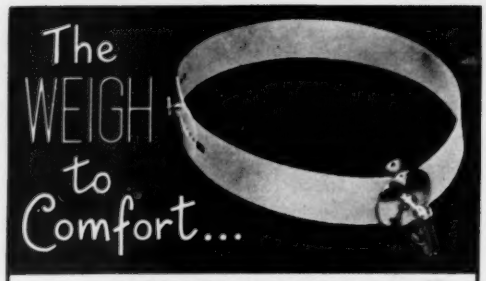

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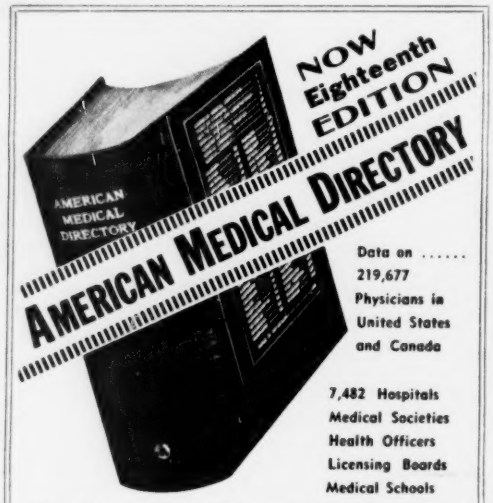

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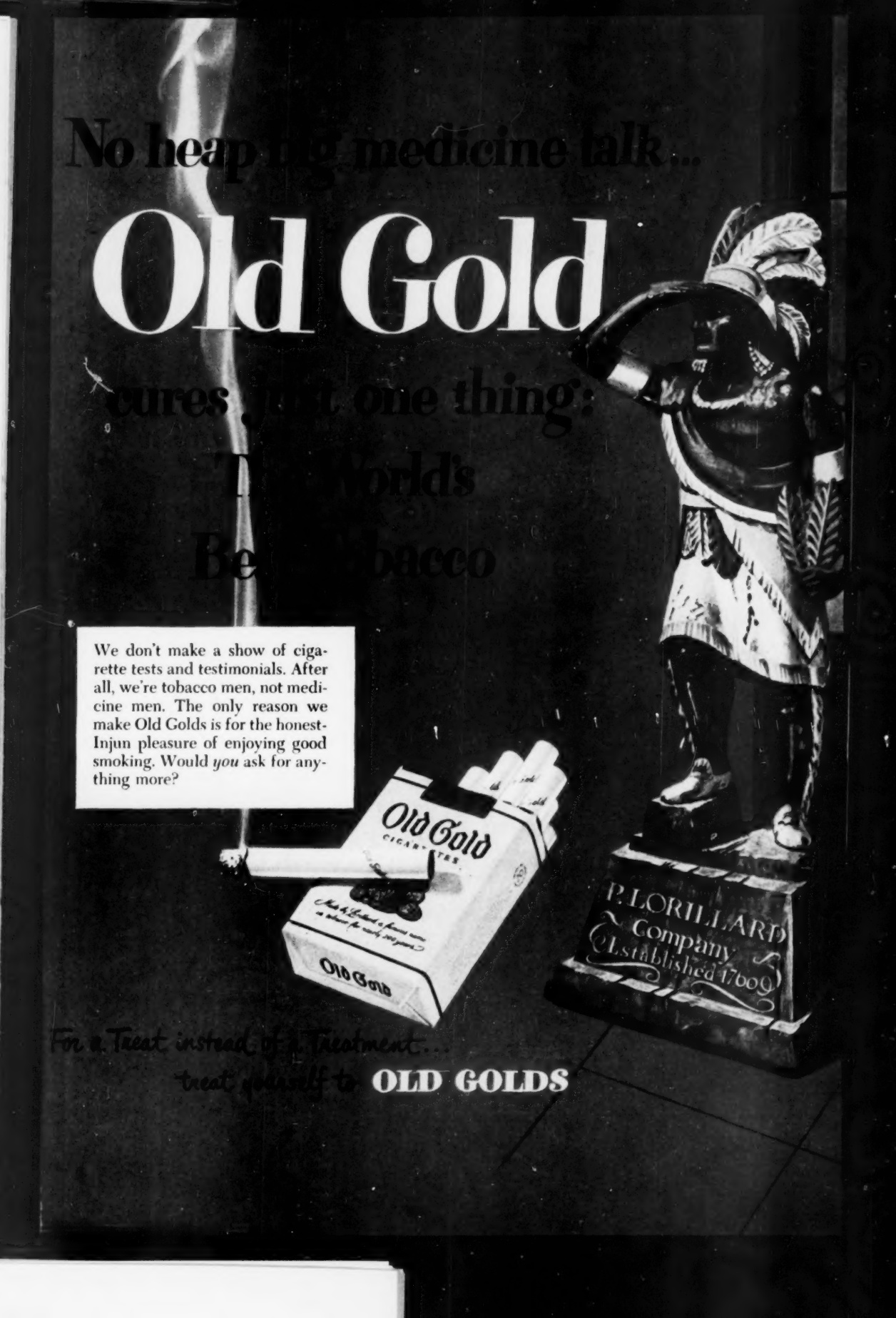
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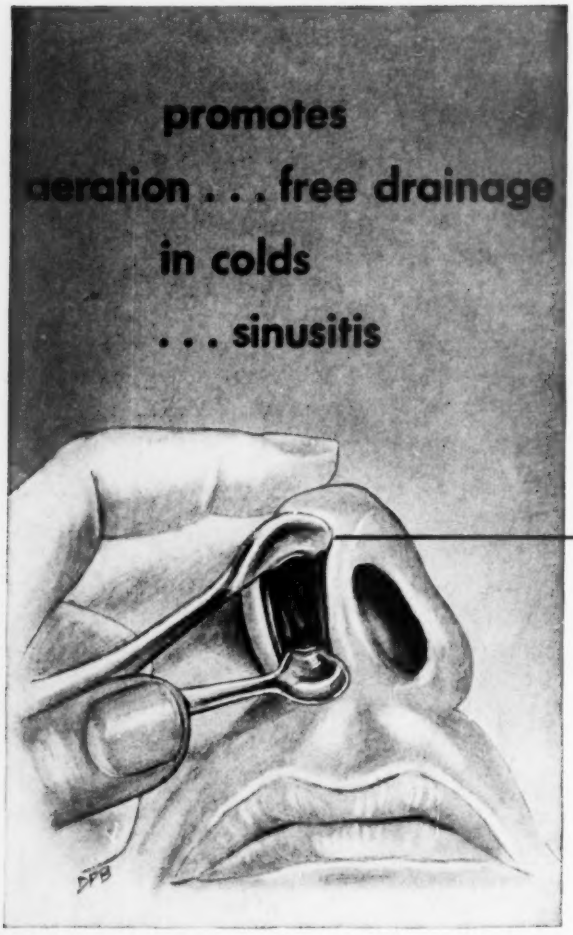
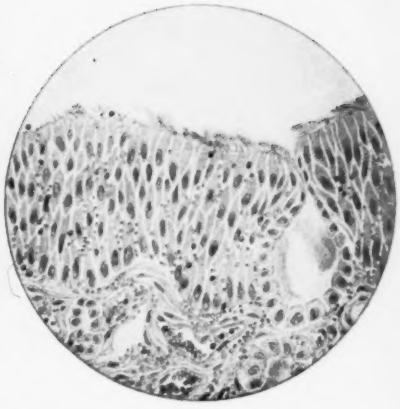
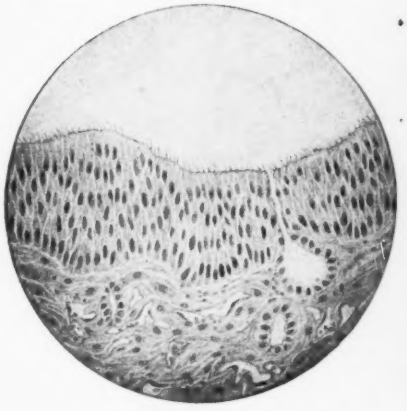




Nasal membrane showing increased leukocytes with denudation of cilia.

Normal appearing nasal epithelium.



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